THE DIABETIC DIET: EDUCATION, COMPLIANCE AND PRACTICAL APPLICATIONS

by

CYNTHIA J. SMITH, B.Sc., S.T.D.

A thesis submitted to the University of Cape Town
in fulfilment of the requirements for the degree of M.Sc. (Med.)

September, 1983

Supervisor: Prof. W.P.U. Jackson, M.A., M.D., F.R.C.P., D.C.H., F.R.S. (S.A.)

To David, Philip and Ilana, in gratitude for their help and understanding.

INDEX

ABSTRACT

The aim of this thesis is to investigate different methods of improving the glycaemic control of diabetic out-patients, within the scope of the author's training both as a therapeutic dietitian and as a teacher. Evidence is presented from the literature, which indicates that high-carbohydrate, high-fibre diets are of benefit in diabetes, that supplements of viscous fibre improve glycaemic control, and that education of the diabetic patient may help to achieve good diabetic control, provided that the patient also complies with all parameters of therapy.

Three main studies have been undertaken:-

(1) An educational project, to investigate the effect of a mass-education programme on compliance and control in diabetic out-patients.
(2) An investigation of the effect of long-term high-fibre diets in diabetic out-patients.
(3) A study of the use of guar gum in the diabetic diet.

In Study 1, a large random sample of patients attending a diabetes out-patient clinic were tested by means of a detailed questionnaire, in order to assess their existing knowledge of the disease. A suitable education programme was then devised and patients were exposed to this in the clinic situation. Another sample of patients was then re-tested with the same questionnaire and statistical analysis was used to assess the effect of this programme on knowledge, compliance and control. Results indicate that, while patients' knowledge scores improved, there was no improvement in dietary compliance and also no significant change in the standard of diabetic control in the clinic population.

In Study 2 we investigated the practical aspects of administering a high-fibre diet to diabetic out-patients in Cape Town, in the light of the reported benefits of diets containing large amounts of dietary fibre (DF) in the control of diabetes. Readily-available, low-cost foodstuffs with a high DF content, were incorporated into suitable, individualised high-fibre meal plans for 10 selected diabetic out-patients. Patients were closely monitored over a period of 9 months, for 3 months of which the high-fibre diet was

prescribed. Various parameters of glycaemic control were recorded and analysed, and the patients' compliance to the new regimen was assessed. Only 3 patients approached the projected fibre intake, but significant negative correlations were found between the dietary fibre increments and both mean plasma glucose and mean serum triglyceride changes. These findings suggest that, were it not for poor dietary compliance, a high-fibre diet might result in significant improvement in diabetic control, and that education and motivation are of prime importance when making major changes to patients' eating habits.

Study 3 investigates the use of guar gum, when incorporated into the diabetic diet in both short- and medium-term studies. This viscous fibre has been shown by workers overseas to be effective in lowering post-prandial glycaemia. In this study a palatable vehicle for the gum, a digestive-type biscuit, was tested for its effect on glycaemic control when incorporated into the usual meal plans of diabetic out-patients, and also against an oral glucose load as a reference standard. It was found to be effective in reducing the post-prandial rise in blood glucose, and in improving glycaemic control, as shown by reduced fasting blood glucose values and decreased 24-hour urinary glucose excretion. The biscuit proved to be palatable and acceptable to patients, and the guar gum was effective in much smaller quantities than have previously been tested. It may therefore prove a valuable adjunct to diabetes therapy.

Results of these studies indicate that compliance to therapeutic recommendations is the crux of achieving good diabetic control. Increased diabetic knowledge alone does not lead to improved diabetic control, and compliance to altered eating habits is difficult to achieve unless prior education and motivation has taken place. The simplest means of achieving better glycaemic control of diabetes appears to be the use of a supplement of viscous fibre, which will improve the glycaemic response to the patients' usual meals.

ACKNOWLEDGEMENTS

The studies described in this thesis were all conducted under the auspices of the Endocrine and Diabetes Research Group of the Department of Medicine, University of Cape Town, and were funded by research grants from the Medical Research Council of South Africa.

As such, I acknowledge with sincere thanks all members of the Research Group for their advice and help, both academic and technical. In particular, I wish to thank Professor W.P.U. Jackson, the supervisor of this thesis, for his invaluable advice, guidance and leadership. The following also deserve special mention:

Dr Les Klaff, the acting head of the Group after Professor Jackson's retirement, for his committment to the concept of "excellent control" and his continual motivation, promotion and support.

Dr Mark Rosman and Dr Naomi Levitt, who helped with much of this research.

Mrs Linda Coetzee and Mrs Susan Abrahams, for their help with all the art-work and illustrations required during the past three years.

Sr Trish Henshilwood and Sr Susan Mee, for their unflagging enthusiasm for the cause of diabetes education.

Mrs Cynthia Woollgar and Mrs Rozanne Simpson, who were always willing to do whatever was required, from typing and preparing rough drafts, to organisation and assistance with the projects.

Mrs Gillian Rubinstein of the Department of Mathematical Statistics, for her detailed analysis of the education project results, and her help and interest in their presentation.

Professor Arthur Vinik, who kindled my interest in guar gum when I was a student dietitian at Groote Schuur Hospital.

Mr Johan Swart of ProSet-Flexoplate, for the expert typing and type-setting of this thesis.

All my friends and members of my family, who tolerated my temporary unavailability when the pressure of work was greatest. This thesis could never have been attempted or completed without the loyalty and support of my husband, who was prepared to spend many nights and week-ends without my companionship, and of my children, who uncomplainingly accepted my preoccupation with these studies.

ABBREVIATIONS

CHO	carbohydrate/s
cm	centimetre
D COMP	dietary compliance
D ED	dietary education
D KN	dietary knowledge
g	gram/s
G COMP	general compliance
G ED	general education
G KN	general knowledge
GTT	glucose tolerance test
HbA_1	glycosylated haemoglobin
HCF	high carbohydrate, high fibre
HDL	high density lipoprotein
HFD	high fibre diet
IDDM	insulin-dependent diabetic patients
kcal	kilocalories
kg	kilograms
kJ	kilojoules
MEDCOMP	compliance with medications
mg/dl	milligrams per decilitre
mg/kg	milligrams per kilogram
mmol/l	millimoles per litre
mmol/24h	millimoles per 24 hours
NIDDM	non-insulin-dependent diabetic patients
p	probability
SD	standard deviation
SEM	standard error of the mean
UV	ultraviolet

Chapter 1

REVIEW OF THE LITERATURE

Section A: Diet and Diabetes

1. HISTORY

"Diet" has always been, and still remains, the cornerstone of diabetic therapy, and the history of dietetic treatment of diabetes covers a period as long as that during which the disease has been recognised. This is illustrated by a dietary prescription in the Papyrus Ebers of approximately 1500 BC, (Jackson and Vinik, 1977) which recommends "a medicine to drive away the passing of too much urine", containing "fresh grits, wheat grains and green lead earth, or sweet beer, honey, grapes and berries". Aretaeus, the Cappadocian, who first gave diabetes its name, wrote in the Second Century AD, "The food is to be milk, and with it the cereals, starch, autumn fruits and sweet wines". (Stowers, 1963). These two examples typify the "carbohydrate-repletion" principle, i.e. replacement of urinary losses with foods rich in sugar.

Perhaps the first detailed diet sheet was prescribed by John Rollo, Surgeon-general of the British Royal Artillery during the late 18th Century. He treated diabetes with a diet composed almost exclusively of meat, preferably rancid and old, together with a pudding made of blood and suet (Mann, 1980). At this stage meat was considered a special remedy for diabetes in European centres, and the principle of carbohydrate-restriction became established. However, the unpalatability of these recommendations precluded them from gaining lasting acceptance.

Other advice during this period included fasting and physical exercise. Later regimens introduced alcohol, or vegetables boiled three times to eliminate the sugar content, or oatmeal cooked with fat and eggs. At the time of the advent of insulin therapy, the main treatments in use were fasting or severe energy restriction, with cautious refeeding until the reappearance of glycosuria was noted. These were popularised by Allen and Joslin, and included the use of a large proportion of fat (75-85% total energy) with severe restriction of carbohydrates in the diet. At this stage diabetes was considered not a disease of total metabolism but rather one

with a specific defect in glucose utilisation.

After insulin became available, diets low in carbohydrates (CHO) and high in fat continued in use. In London in 1931, the accepted hospital diet provided 15% of the energy intake as CHO with 17% as protein and 68% as fat. (Mann, 1980). Carbohydrate restriction remained in vogue for many years, while other approaches were investigated.

2. HIGH-CARBOHYDRATE DIETS

The initial post-insulin diets were thus much higher in animal fat and cholesterol than those taken by persons without diabetes and in 1928 Joslin proposed that high-fat diets might aggravate the atherosclerosis so prevalent in diabetic patients. (Anderson, 1980.)

In addition, other American studies suggested that high-fat diets, which had been used with CHO in proportions to obviate "acid-body formation", were not well absorbed or tolerated by the patient (Richardson, 1929). It was found that the amounts of fat and CHO in the diet could be transposed and still have the patient "sugar free" even when the dietary CHO was raised from 57 to 150 or 200 g per day. The patients also adhered more closely to these diets and felt better. Newer high-CHO diets advocated the use of bread, oatmeal, potato, milk, fruit and even sucrose, with a lower fat: CHO ratio, and it was reported that low caloric diets combined with higher CHO content satisfied patients better. (Sansum, 1926).

Improved techniques of dietary analysis (Lawrence and McCance, 1929) indicated that the carbohydrate content of foods was actually higher than previously calculated and could be divided into "available" and "unavailable" types. With this knowledge it was permitted to allow larger amounts of green vegetables and it was no longer deemed necessary to boil them three times, due to their low content of available carbohydrate. The more liberal diets now contained fruits as well, and allowed up to a 60% increase in the carbohydrate content, especially with the encouragement of foods high in unavailable carbohydrate. The urines of patients on these new allowances contained less "acetone bodies" than before.

The careful clinical observations of Rabinowitch (1935) supported the mounting evidence for improved diabetic control on higher carbohydrate

allowances. Insulin doses recorded on diets providing 54% of energy as CHO were less than one third of those required on low-carbohydrate diets. Rabinowitch also documented the improvement of his patients with caloric restriction and weight loss. The tendency to abandon the high-fat, low-CHO diets for diabetes was supported by studies in India (Singh, 1955). Here 80 insulin-sensitive diabetics were treated with low fat, high CHO optimal energy diets, in order to achieve normal or slightly below normal weight, and a progressive reduction in insulin requirements was observed, with improvement in glucose tolerance tests performed 18-26 months later.

The classical studies of Himsworth demonstrated that glucose tolerance in normal subjects improved on a high carbohydrate, low fat diet (Himsworth 1933 and 1935) and by deductive reasoning that the increase in carbohydrate was the governing factor for this improvement. In diabetic subjects he showed that a standard dose of glucose produced less hyperglycaemia on a high carbohydrate diet than on a high fat diet and that injection of a standard dose of insulin was more effective on the high carbohydrate diet. He later suggested that diabetics could be classed as "insulin-sensitive" or "insulin insensitive". Those of the former type responded to an increase in dietary carbohydrate with either no increase or a decrease in glycosuria, due to an improved tissue sensitivity to insulin (Himsworth, 1939), while insulin-insensitive patients showed impaired glucose tolerance, and an aggravation of hyperglycaemia and glycosuria on a high carbohydrate diet. He postulated that in sensitive individuals "the effect of insulin in depressing the blood sugar is proportional to the height of the resting blood sugar level". Moreover, temporary insulin insensitivity could be induced by "carbohydrate starvation" (Himsworth, 1949).

The variable response of the body to a glucose tolerance test depending on the state of carbohydrate repletion or depletion prior to the test, was known at this time. Sweeney (1927) had suggested a standardised antecedent diet in order to eliminate the variability of response, while Gibson (1929) had demonstrated that 2-3 days of high sugar ingestion, coupled with increased insulin as required, resulted in improved tolerance upon resumption of the usual diabetic diet.

Thus the carbohydrate-restriction principle was challenged by clinical experience and scientific studies. As a result, there was a gradual increase of carbohydrate content in diabetic diets up to 35% or 40% of energy content by the 1960's. Further reports continued to support this modest level of carbohydrate restriction. Brunzell, Lerner, Hazard et al (1971) showed that increasing the carbohydrate content of the diet, provided in liquid form by dextrose or dextrimaltose, from 45% to 85% of the dietary energy, produced a fall in fasting plasma glucose in normal subjects as well as in "mild diabetics" (non-insulin-dependent patients with mean fasting blood glucose of ± 120 mg/dl). The fasting serum insulin also showed a fall, and glucose tolerance improved after 10 days of high-carbohydrate feeding. The authors suggested the mechanism for the decrease in fasting glucose levels and for the improved glucose tolerance was that the high level of carbohydrate increased the sensitivity of the tissues to insulin, since the insulin responses to an oral glucose load did not change significantly. A similar conclusion was reached by the same authors in 1974 (Brunzell, Lerner, Porte et al, 1974) whereby they showed a significant decrease in the fasting plasma glucose of patients on insulin or oral agents, when they were fed formula diets containing 85% carbohydrate and no fat, compared with their response to diets containing 45% carbohydrate and 40% fat. Only untreated diabetics fared worse on the fat-free high carbohydrate diet. The diets in this case were calculated to be weight-maintaining.

This was in contrast to earlier work (Rudnick and Taylor, 1965) where low-calorie, low-carbohydrate diets resulted in improved glucose tolerance in "mild obese diabetics". The authors suggested that the capacity of the pancreas to secrete insulin improves following dietary management to lower the blood glucose level. A retrospective study by Jackson and co-workers (1972) supports this view of the importance of carbohydrate restriction, as does a prospective study by Wall, Pyke and Oakley (1973). Another aspect of therapy for obese diabetics was highlighted by Grey and Kipnis (1971), who showed that the basal plasma insulin decreased on refeeding to an isocaloric high carbohydrate diet, despite continued weight loss. The insulin response to an oral glucose tolerance test decreased likewise on a low carbohydrate diet, and

increased on a high-carbohydrate intake.

Although the results of these studies appear to be at variance with one another, they do in general confirm Himsworth's original observations, and conclusions can be drawn as follows:-

(i) Increase in dietary carbohydrate improves glucose tolerance in treated subjects.
(ii) Low-carbohydrate, weight-reduction diets improve glucose tolerance in mild obese diabetic patients.
(iii) Plasma insulin levels in response to high carbohydrate diets may rise after weight loss, due to recovery of the pancreas to a certain degree, but this is probably accompanied by increased tissue sensitivity to insulin.

In other more recent studies of all categories of diabetics (Anderson, 1980, and Kiehm, Anderson and Ward, 1976), increasing carbohydrate content of the diet (as solid food) to 75% of the calories, produced lower fasting plasma glucose and improved glucose tolerance tests, as Brunzell had found. In addition, subjects were able to reduce or discontinue their doses of insulin. Similarly, restriction of carbohydrate to 20% of the calories produced a significant deterioration in glucose tolerance. Thus later studies continued to support Himsworth's theories and the concept of high-carbohydrate dietary therapy for treated or less severe diabetics. It has been suggested that high-carbohydrate diets, besides increasing exogenous and endogenous insulin sensitivity, may induce enzymes in the liver or jejenal mucosa which metabolise glucose (Kiehm, Anderson and Ward, 1976; Anderson, 1980).

3. MODIFIED FAT DIETS

Concurrently with the mounting evidence for improved diabetic control on high carbohydrate diets, experimental and epidemiological data was accumulating regarding the increased risk of premature cardiovascular disease in diabetics, as well as for improved treatment of the hyperlipidaemic patient. Again this had been initiated in the 1920's by Himsworth and Joslin, who had suggested that the high-fat diets were detrimental to the metabolic abnormalities of diabetes. Elevated plasma

cholesterol and triglyceride levels are common findings in diabetic patients (Simpson, Mann, Hockaday et al, 1979a) and are known to predispose to the development of macrovascular disease both in the general population (Carlson and Bottiger, 1972) and in diabetics (Medalie, 1979). Diets low in fat, with an increased proportion of poly-unsaturated fats, have been shown to lower cholesterol levels in non-diabetic subjects, (Miettinen, Turpeinen, Karvonen et al, 1972), and epidemiological evidence abounds that such diets are associated with a reduced incidence of heart disease. (Shekelle et al, 1981; Kato et al, 1973; Oliver, 1981).

Several studies have specifically investigated the effect of low-fat and/or low-cholesterol diets on diabetic patients. Insulin-dependent patients, put on a low-cholesterol, high-carbohydrate diet for one year, showed a significant decrease in their lipid levels, even without an appreciable decrease in weight. (Stone and Connor, 1963). Similar results have been shown in both insulin-dependent (IDDM) and non-insulin dependent (NIDDM) subjects given a high-carbohydrate low-fat diet (Kiehm, Anderson and Ward, 1976).

The realisation that low-fat diets with appropriate modification of fatty acid composition could be combined with the increasingly accepted high-carbohydrate diets, led workers in Oxford to compare the standard diabetic diets (40% energy from carbohydrate) with a high-carbohydrate (54% energy), modified-fat diet. (Hockaday, Hockaday, Mann et al, 1978). Newly diagnosed untreated overweight diabetics were placed at random on either of the dietary regimens, to assess their response in respect of circulating metabolites. After one year, the fasting plasma glucose levels and body weights showed a similar decrease on both diets, but the plasma cholesterol had decreased only in those subjects on the modified fat diet. It is interesting to note that no special emphasis was laid here on the type of dietary carbohydrate supplied, and that both regimens were energy-restricted according to the degree of overweight. It was concluded that energy restriction to achieve weight reduction, was the single most important factor in glycaemic control of newly diagnosed, non-insulin-dependent patients.

In a later study (Simpson, Mann, Eaton et al, 1979b) a high-carbohydrate, modified-fat diet providing 60% energy from digestible and

non-digestible carbohydrate was used for treatment of maturity-onset diabetics, with stipulated inclusion of cereal foods, wholemeal bread, tuberous vegetables and fruit, and restriction of mono- and di-saccharides. Here mean basal and pre-prandial plasma glucose values as well as glycosylated haemoglobin decreased, and the fasting plasma cholesterol showed a concommitant fall. The authors postulated that the fall in basal and pre-prandial blood glucose was mainly due to the effect of the digestible carbohydrate on the blood glucose levels. It was also found that the triglyceride levels were not affected by the high-carbohydrate diet, thus allaying fears that high-carbohydrate diets would aggravate hypertriglyceridaemia.

It appears from these studies and others that high-carbohydrate diets of the correct constituents, with restriction of mono- and di-saccharides, do not permanently elevate serum triglycerides when such diets are maintained for long periods of time in normal subjects (Antonis and Bersohn, 1961) and in diabetics (Stone, 1963). Earlier studies which suggested that this elevation might occur, has used simple sugars such as dextrins or lactose as their carbohydrate source (Farquhar, Frank, Gross et al, 1966). The initiating factor for the hypertriglyceridaemic response to such a high-carbohydrate diet was postulated to be hyperinsulinaemia, which may be alleviated by the increased tissue sensitivity to insulin produced by a high-carbohydrate diet, as discussed above.

Conflicting evidence of the effect of high-carbohydrate low-fat diets in non-obese diabetics, was produced by Weinsier, Seeman, Herrera et al (1974). Here no differences in diabetic content or lipid metabolism were found whether patients were on a 60% or 40% carbohydrate diet. However, both test groups in this study had been given advice on dietary fat. The Oxford group subsequently investigated the effect of their high-carbohydrate modified fat diet on insulin-dependent subjects, with further positive findings (Simpson, Mann, Eaton et al, 1979c). After six weeks on the test diet, the average total daily insulin requirement was reduced significantly, while the mean basal plasma glucose concentrations, total plasma cholesterol and LDL cholesterol all fell significantly. Apart from nocturnal hypoglycaemia, all aspects of carbohydrate and lipid metabolism thus improved.

The importance of the low-fat dietary approach in diabetes therapy for NIDDM patients is emphasised anew by a recent randomised study by de Bout, Baker, St. Leger et al (1981), when it was found that a low fat diet group showed significantly greater decrease in body weight and serum cholesterol, with no deterioration in diabetic control. Simultaneously with the reduction of dietary fat intake achieved in 6 months, the subjects also increased their carbohydrate intake, although they had not been specifically instructed to do so. Thus benefits for both metabolic abnormalities of diabetes were achieved with only one dietary emphasis.

4. HIGH-FIBRE DIETS.

With the evolution of the dietary fibre hypothesis by Burkitt and Trowell in the early 1970's, the work on high-carbohydrate diabetic diets gained more impetus as well as a new direction. Since epidemiologically an overconsumption of fibre-depleted starchy foods was postulated to be an aetiological factor for diabetes mellitus (Wapnick, Wicks, Kanengoni et al, 1972; Walker, 1970; Trowell, 1973, 1975) besides its involvement in the so-called Western diseases of the heart and bowel (Walker, 1961), the converse was also postulated, i.e. that high-carbohydrate, high fibre foods would be beneficial in diabetes therapy.

(a) Types of Dietary Fibre

Dietary fibre was defined as "the remnants of plant cell walls which are not hydrolysed by the digestive enzymes of man" (Trowell, 1973), and this definition was later expanded to include "the plant polysaccharides and lignin" (Trowell, Southgate, Wolever et al, 1976). It thus incorporates storage polysaccharides, gums and mucilages, as well as structural forms of fibre. As such it is not a chemical entity but a complex mixture of substances and will vary according to the type of food or habitual diet consumed.

Plant fibres may be divided on the basis of water solubility into insoluble and soluble types. The insoluble fibres include cellulose, lignin and many hemi-celluloses, while the soluble fibres include pectins, some hemi-celluloses, gums, mucilages and storage polysaccharides (Anderson and Chen, 1979).Total dietary fibre, measured by the method of Southgate

(1976) includes both insoluble and soluble types, and thus gives higher values than the method of van Soest and Robertson (1977), which was referred to as analysis for crude fibre.

Cellulose is the best known and most widely distributed component of the plant cell wall. It represents about 25% of the fibre content of many grains, fruits and vegetables. Lignin, which is not a carbohydrate in structure, is likewise a component of the cell wall, and it may account for approximately 10% of total fibre of grains, vegetables and fruit. Hemi-celluloses are polymers of pentose and hexose sugars, and may be associated with cellulose in the cell-wall. They constitute 50-70% of the fibre content of grains and vegetables. Pectins are also structural components of the cell wall, consisting of branched polymers of galacturonic acid, linked to other carbohydrate units such as arabinose or xylose.

The main source of pectins in the diet is fruit, and they are completely digested by bacteria in the colon. Gums and mucilages have similar biochemical and physiological properties to the pectins and hemicelluloses, although they are not components of the cell wall. Storage polysaccharides are usually mixed with starch in the endosperm of leguminous seeds, but are not digested in the small intestine (Anderson, Midgley and Wedman, 1979).

(b) Properties and Effects of Fibre

(i) Water absorption

Plant fibre in general and cellulose in particular is capable of absorbing large quantities of water in the gut. Some fibres form gels with water; these include pectins, gums, mucilages and storage polysaccharides. Once hydrated, they form a type of molecular sieve which could alter digestion and absorption of nutrients.

(ii) Altered intestinal transit time

In general, foods rich in insoluble fibres (e.g. whole grain products) decrease transit time and increase faecal bulk, while soluble fibres (e.g. gums and pectins) form gels which delay gastric emptying and increase transit time, while having no effect on faecal bulk.

(iii) Binding

Cations such as iron, magnesium, calcium and zinc, may be bound to acid sugars in the fibre complex, as may organic substances such as bile salts. This may result in short-term negative mineral balance (Moynahan, 1977) but over longer periods adaptive responses probably enhance the efficiency of absorption, and long-term studies have revealed no deficiencies of minerals or vitamins in diabetics on high-fibre diets (Anderson, Ferguson, Karounos et al, 1980).

(iv) Energy intake

The energy value of a high fibre diet may also be overestimated, since the apparent digestibility of other nutrients may be reduced with a higher intake of fibre (Southgate, 1973). If intestinal transit time is reduced, then there may be less time for digestion and absorption, and faecal excretion of energy, fat and protein may increase (Kelsay, Behall, and Prather, 1978). This, however, may be offset by the absorption of volatile fatty acids produced by bacterial fermentation of fibre in the colon.

(v) Carbohydrate metabolism

The effect of dietary fibre ingested with carbohydrate was elegantly demonstrated by the studies of Haber (1977), who compared the influence on plasma glucose and insulin of whole apples (intact fibre) with apple puree (disrupted fibre) and with apple juice (depleted fibre). All feedings contained 60g carbohydrate, but their effect on plasma glucose varied. Intact fibre produced a smaller rise in plasma glucose and a return to basal level with no rebound fall, while ingestion of apple juice resulted in an earlier glucose peak and a rebound fall below fasting levels, simultaneously with a larger serum insulin response. The meal of CHO with disrupted fibre caused an intermediate effect. This experiment indicated that physical disruption of fibre alters physiological properties and supported the fibre-depletion theory of the aetiology of diabetes mellitus. Similar effects were noted in diabetics (Lenner, 1976) where meals with the highest proportion of oligosaccharides given

as sucrose, fructose or lactose caused symptoms of hypoglycaemia.

(c) *The use of guar gum*

Various types of fibres were tested for their efficacy in improving glucose tolerance, both in normal subjects (Munoz, Sandstead and Jacob, 1979; Wahlquist, 1979) and in diabetics (Jenkins, Wolever, Leeds et al, 1978a; Miranda and Horwitz, 1979; Monnier, Pham, Aguirre et al, 1978). Guar gum was found to be the most effective, while wheat bran seemed to produce no improvement. Guar gum is a storage polysaccharide found in the endosperm of the Indian cluster bean, *Cyamopsis tetragonoloba*, and is used commercially as an emulsifying and thickening agent in various foods. Its hygroscopic properties result in gel formation both in foods and in the small intestine. The mechanism of its effect on decreasing post-prandial glycaemia is thought to be its viscous nature, which would delay gastric emptying and also small intestinal absorption. (Holt, Heading, Carter et al, 1979; Eastwood and Kay, 1979).

The W.H.O. Expert Committee on Food Additives originally (1970) assigned to guar gum a temporary acceptable daily intake of 0-125 mg/kg body weight, but later the Committee stated that it was no longer deemed necessary to establish a safe level of intake, as guar gum did not represent a hazard to health (W.H.O., 1975). No toxicological, histopathological or teratogenic effects were found in animal studies, and the galactomannan showed no evidence of hydrolysis by mammalian intestinal enzymes (rat and human). The pioneer in studies on guar gum, Jenkins, fed test meals containing 14-16g guar, mixed in fruit juices, soups or other drinks to normal and diabetic subjects, and showed that decreased post-prandial glycaemia resulted. (Jenkins, Goff, Leeds et al, 1976; Jenkins et al, 1977a and 1977b). He also administered guar to diabetic patients in metabolic ward studies and as supplements in their normal home diets, for 5-7 day periods, in aliquots totalling 25g per day. Urinary glucose excretion fell dramatically (Jenkins, Wolever, Hockaday et al, 1977 b), and some patients found it necessary to reduce their insulin dosage temporarily.

Due to problems of administering the viscous guar in drinks, an attempt was made to produce guar bread (Apling, Leeds, Wolever et al, 1977) and also crispbread (Jenkins, Wolever, Nineham et al, 1978 b). Here Jenkins

used 14-26g guar per day, in home and metabolic ward studies. In order to prevent hypoglycaemic episodes, insulin was reduced by a mean of 8 units and carbohydrate increased by 40-50g daily, prior to the test period. He showed a reduction in urinary glucose loss and a relative fall in fasting blood glucose concentrations, as well as a "carry-over" effect of guar on urinary glucose for a subsequent 2 days after the guar treatment. This extended influence of guar on the glycaemic response was also shown by Jenkins, Wolever, Nineham et al, (1980 a) to exist with a second glucose tolerance test 4 hours after a glucose load combined with 22,3g guar, and he suggested that it might be due to a slower rate of CHO absorption in response to the initial glucose and guar level, and thus less stimulus for ketone body formation. Because of this, glucose would be taken up more readily by peripheral tissues during the second load. Jenkins also emphasised the importance of fibre viscosity in improving glucose tolerance, and stressed that fibre should be mixed intimately with food in order to simulate a situation analagous to that of unprocessed foods. Inadequately mixed guar has been shown to be ineffective (Cohen et al, 1980; Williams et al, 1980; Kuhl et al, 1983).

The mechanism of action of guar may also be mediated through gut hormones, (Morgan, Goulder, Tsiolakis et al, 1979), as well as through delayed gastric emptying. In glucose tolerance tests on patients with autonomic neuropathy, guar was shown to have no effect (Levitt, Vinik, Sive et al, 1980), and it was argued that with already delayed gastric emptying in this condition, guar could have no further influence.

The artificial pancreas has confirmed a small but significant reduction in 24-hour insulin requirements during guar supplementation (20g/day) (Christiansen, Bonnevie, Svedsen et al, 1980), although no effect was shown on the "mean amplitude of glycaemic excursions". Here again, this could have been due to inadequate mixing of guar with food, as it was taken with water before meals or snacks.

Supplementation of meals with guar gum, then, has been shown to improve glycaemic control in general, although it does not produce weight loss in poorly-controlled NIDDM (Ray, Mansell, Knight et al, 1983). It does, however, improve the lipid pattern of both IDDM and NIDDM (Jenkins, Wolever, Taylor et al, 1980 b) and may increase tissue sensitivity

to insulin in NIDDM (Aro, Uusitupa, Voutilainen et al, 1981). Long term use of guar supplements (14-26g per day for 6-12 months) does not seem to produce any mineral or trace-element deficiency (Jenkins, Wolever, Taylor et al 1980 b). The major problem with guar supplementation, however, is its unpalatability and unpleasant side-effects. 16g in orange juice was documented to produce an abdominal upset 5-8 hours after ingestion (Holt, Heading, Carter et al, 1979), and particularly in the large doses used by Jenkins, guar would cause diarrhoea, flatulence and abdominal distention.

(d) Other high-fibre studies

Simultaneously with the studies on guar gum as a fibre supplement, other workers were approaching the problem from a different view-point – that ordinary high-fibre foods could be used to improve diabetic control. It had already been shown that raising the CHO content of the diet from 40% to 60%, had no adverse effect on diabetic control (Weinsier, Seeman, Herrera et al, 1974), although if carbohydrates were provided in the refined or liquid formula form, they tended to elevate serum triglycerides (Reiser, Hallfrisch, Michaelis et al, 1979).

Miranda and Horwitz (1978) reported the use of bread and fruit to provide 20g crude fibre per day for 10 days to 8 IDDM patients. Here, with carbohydrate providing 42% of the energy, the mean plasma glucose decreased significantly on the high fibre diet (HFD) and hypoglycaemic reactions occured more frequently. Anderson and co-workers in Kentucky also tested the fibre hypothesis in terms of high carbohydrate, high fibre (HCF) diets. He provided 70% CHO diets containing 64g dietary fibre to obese and non-obese men both in metabolic ward studies (Anderson and Ward, 1979) and at home (Anderson and Ward, 1978). The fibre in these diets was provided by cereals, vegetables, legumes and fruit, taken as whole foods rather than as supplements. Significant reductions in requirements for insulin or oral agents were achieved and in some cases insulin or oral agents were withdrawn totally. Fasting plasma glucose and serum triglycerides were significantly lower on the HCF diet. Patients were discharged on a maintenance diet of 55-60% CHO and those who followed this HCF diet maintained their lower insulin dose, while those

who returned to the control diet resumed their previous dosage levels. Anderson has been able to maintain some patients on the 55-60% CHO diet as outpatients for up to 3 years, with no deterioration in diabetic control. Similar improvements have been noted in IDDM patients (Anderson, 1980) and he has demonstrated increased insulin binding to monocytes on the HCF diets. This has recently been confirmed by Pedersen, Hjolland, Lindskov et al, (1982) who showed that monocyte insulin receptor binding is positively correlated with glucose tolerance and with *in vivo* insulin action.

Studies in Oxford supported Anderson's work. Simpson's work on modified-fat diets for NIDDM patients has already been discussed (Simpson, 1979b). He subsequently tested IDDM patients as well, on a high-carbohydrate diet using cereal and vegetable sources of fibre (Simpson, Mann, Eaton et al, 1979c). Several parameters of diabetic control improved significantly when patients were on the HCF part of this cross-over study. An extension of his work, possibly based on Jenkins' success with his guar studies, used leguminous (viscous) fibre (kidney, haricot and butter beans) in addition to cereal fibre (wholemeal bread) in both IDDM and NIDDM subjects for 6 weeks, and again several indices of diabetic control improved (Simpson, Simpson, Lousley et al, 1981). Here 64% of the 150g fibre per day was provided by legumes, and the extent of the dietary change required is shown by the fact that 73% of the dietary energy in this study was obtained from bread and legumes.

Workers in Naples (Rivellese, Riccardi, Giacco et al, 1980) took advantage of differences in regional taste to provide fibre from legumes including chick-peas and lentils and other vegetables such as fennel, artichokes, egg-plant and bean sprouts. The effect of fibre was investigated by providing 3 types of diet: 53% CHO low fibre, 53% CHO high fibre and 42% CHO low fibre. Post-prandial glycaemia and the mean daily blood glucose levels were significantly lower when subjects (IDDM & NIDDM) were on the 53% CHO high fibre diet. In contrast to Simpson's work, all diets used here were similar in fat content and in the ratio of poly-unsaturated fatty acids. Total cholesterol and LDL cholesterol decreased on the HCF diet and serum triglycerides were not elevated. Again, insulin dosage was reduced when IDDM were on the HCF diet.

This study did not indicate any benefit of an increased carbohydrate intake, but clearly showed the advantage of high fibre foods.

The particulate form of fibre (i.e. bran) was used in other studies with varying results. In short-term studies, bran retarded intestinal glucose absorption only in NIDDM treated with oral agents (Hall, Bolton and Hetenyi, 1980) while in patients treated by diet alone, the insulin response was reduced, and its effect potentiated by bran. Here, as in longer-term studies (Bosello et al, 1980; Monnier et al, 1981) the mechanisms remain obscure, although glucose tolerance appeared to improve. In general, however, the effect of viscous fibre seems more impressive than that of bran.

An important illustration of how naturally available foods can be used to incorporate fibre into the daily diet, was given by Kinmonth, Angus, Jenkins et al (1982). The authors chose the families of 10 children in the Oxford area, and changed their traditional low fibre, low CHO diet to either a high CHO refined diet or an unrefined high CHO diet. Each diet was identical as regards the energy, fat and protein content. The low fibre diet had 10g fibre/1000 kcal, and the CHO content consisted of ⅔ as white bread and ⅓ as fruits, vegetables (processed) and milk. 50% of the CHO on the HFD was provided by wholegrain cereals, and 50% by fresh fruit and vegetables and dried beans. The children and their families were put on each diet for a 6 week period. It was found that all blood glucose levels were lower on the unrefined diet, even pre-breakfast, and that the mean 24-hour blood glucose and urinary glucose values were also significantly lower. However, most children found it unacceptable to remain on the restricted amounts of meat and cheese after the study had ended, for longer than three months. Six months later, nevertheless, all children had changed their eating habits to include significantly increased amounts of dietary fibre and CHO. Hopefully this would benefit them in the long-term.

5. COMBINED EFFECT OF CARBOHYDRATE AND FIBRE

It has been postulated by several authors that there is a beneficial effect of digestible CHO on its own, i.e. that diabetic control may not only be improved by dietary fibre but by a high intake of digestible CHO in the

diet. Simpson, Carter, Lousley et al (1982) suggested that complex digestible CHO (as white bread for example) would benefit the basal blood glucose levels, while fibre would blunt the post-prandial glucose rise. In this study, however, all the patients were well-controlled at the commencement of the trial. A high-CHO diet may not be beneficial to newly diagnosed or poorly-controlled patients. Caloric restriction and weight-reduction are still the most important means of improving glycaemic control in NIDDM (Hoffman, Fineberg, Howey et al, 1982). However, Simpson's theory does explain the apparent lack of effect of high-fibre diets on fasting blood glucose reported by Miranda and Horwitz (1978) – here the CHO content was only 40% of the calories. Jenkins, Wolever, Bacon et al (1980c) tested this concept by giving guar supplements in diets with varying levels of CHO intake, and found that only when CHO formed more than 40% of the calorie intake, was there a reduction in glycosuria. The lack of effect of guar in the glycaemic control of pregnant IDDM patients, shown by Kuhl, Pedersen, Hornnes et al (1983) could also be due to the low CHO diet used in this study (less than 40% energy).

Thus it is possible that fibre and carbohydrate act synergistically in achieving diabetic control, that serum lipid patterns are improved by the HCF diets which also necessitate reduction in total fat content by virtue of the increased contribution of carbohydrate to the energy value, and that viscous fibres are most beneficial in reducing post-prandial glycaemia.

6. CURRENT DIETARY RECOMMENDATIONS

In the light of these findings, the Food and Nutrition Committee of the American Diabetes Association (1979) published new dietary recommendations for diabetics. These stated that "some liberalisation of carbohydrate intake is recommended", preferably as complex carbohydrate (starch associated with fibre) and as a replacement for some of the fat. Therapy for the obese NIDDM should still be based on weight reduction.

Recent publications of the British and Canadian Diabetes Associations include proposals which concur with the American ones regarding fat and carbohydrate content. (Arky, 1982). The restriction of simple sugars is

re-emphasised, since high-carbohydrate intakes should not be interpreted as leeway to ingest "42% of calories as Danish pastry" (Reaven, 1981). The proportion of carbohydrate calories in the diet range from 50-60% (American) to 50-55% (British) and minimum 45% (Canadian). Thus the recommendations are remarkably similar, and accept in principle that a higher CHO intake is "consistent with the goal of achieving normal blood sugars."

7. RECENT DEVELOPMENTS.

It has been known for some time that different forms of CHO-containing foods produce different effects on post-prandial glycaemia, even when given in equivalent amounts. Thus Swan, Davidson, Albrink et al (1966) showed that the plasma glucose response to starch was lower than that to glucose or sucrose. Here, however, the starch was given in solid form as bread and water, while the glucose or sucrose were given in solution as drinks. This may have affected the rate of gastric emptying and absorption of the digested monosaccharides, and thus the peak plasma concentrations of glucose.

Crapo and colleagues (1976) eliminated these problems of comparison by administering equivalent glucose loads as glucose, sucrose and starch, both as drinks (pure CHO) and as meals (CHO in combination with protein and fat). Simple CHO were found to produce a greater and faster rise in post-prandial glucose and insulin responses than complex CHO, while meals produced a lower plasma glucose response than drinks. When the same authors tested cooked potato and rice instead of soluble starch, potato produced a greater glucose and insulin response than rice, but both were less than that of the glucose drink. Bread was found to have a response intermediate between potato and rice or corn (Crapo, Reaven and Olefsky, 1977) in normal volunteers and in individuals with impaired glucose tolerance (Crapo, Kolterman, Waldeck et al, 1980).

As early as 1937 it was suggested that dietary recommendations should be based on biological responses to foods rather than on their chemical content (Crapo and Olefsky, 1983) since the analysed CHO content did not correspond with the subsequent blood sugar response, and this was re-asserted by Schauberger, (1977) who found that fruit and peas produced

a lower plasma glucose response than bread.

The insulin response to test meals was also studied. Sucrose was found to elicit a greater response than glucose (Crapo, Reaven and Olefsky, 1976) and than rice or corn (Coulston, Greenfield, Kraemer et al, 1980), in normal subjects. In NIDDM patients, Ionescu-Tirgoviste (1983) found that fructose and lactose produced lower blood glucose increases than glucose, and that honey and carrots gave the lowest blood glucose increase. Rice and potato had slower absorption than apples, which produced a rapid increase and a peak in blood glucose at 30 minutes. This seems to confirm the standard view that simple sugars are more rapidly absorbed than complex CHO and therefore aggravate hyperglycaemia in diabetics.

It now appears, however, that there is a spectrum of biological responses to various simple and complex CHO and that no clear-cut division exists. Jenkins and his co-workers have tested equivalent portions of CHO given as dried legumes, cereals, breads, pasta, biscuits and root vegetables, to normal volunteers (Jenkins, Wolever, Taylor et al, 1980 d) and to diabetic subjects (Jenkins, Wolever, Jenkins et al, 1983a). His results seem to indicate that there is no effect of fibre, since white and wholemeal bread or rice elicited similar blood glucose responses (Jenkins, 1981a), but rather that the form of the food is important, in that it determines its accessibility to digestive enzymes in the gut. Pasta, with its more compact nature, produces a lower glycaemic response than bread or rice. This supports the findings of Haber, where depletion or disruption of fibre in apples was shown to affect the blood glucose response, and also the work of Collings, (1981) who showed that cooked starch produced a greater glucose response than raw starch. Jenkins suggests that disruption of the normal relationship of starch and fibre, as in milling of wheat products, may reduce the effect of fibre on post-prandial glycaemia, and that wheat starch may be more beneficial if fed as pasta than as bread. The importance of food form on absorption is also shown clearly in experiments done by O'Dea (1980), where ground rice produced a higher glucose and more rapid insulin response than whole rice, even when brown rice (high in fibre) was used. Presumably here too the surface area exposed to digestive enzymes is the key factor in determining post-prandial

metabolic responses.

An *in vitro* method of testing the rate of enzymatic digestion has been developed (Jenkins, Wolever, Taylor et al, 1980e) and found to correlate well with the glucose response *in vivo*. It was found that leguminous seeds, lentils and soya beans in particular, liberated significantly less CHO measured as glucose *in vitro* when compared to bread. Thus it is postulated that, apart from the effect of viscous fibres on gastric emptying, events in the small intestine may determine the rate of release of absorbable oligosaccharides. Legumes are also rich in inhibitors of amylase activity and thus of CHO digestion. 75% of sugars have been shown to be absorbed in the proximal 70cm of small intestine (Jenkins, Ghafari, Wolever et al, 1982a), but the total length of small intestine available for absorption is approximately 6-7 metres. Thus slowly-released CHO may be efficiently and more gradually absorbed, causing a flatter, more prolonged blood glucose response. A different gut endocrine response may also be provoked by a more distal site of absorption of glucose in the small intestine, resulting in long-term improvement in glucose tolerance and/or insulin requirement.

Some foods liberate free sugars readily, without enzymatic digestion. Southgate (1978) has stated that all foods are biological mixtures and not simple entities. Thus their composition is not constant, and in particular the CHO content could vary in the amount of starch and free sugars, according to the degree of ripeness, length of storage, cooking, season, etc. 55% of the CHO in sweet-potato may be taken as starch, for example, while the rest consists of free sugars.

Jenkins (1982) has devised a "glycaemic index" to indicate the effect of various foods on blood glucose. This is a ratio of the area under the 2-hour food glucose curve to the area under the 2 hour GTT glucose curve, and resulted from *in vivo* studies of 50g portions of CHO given as different foods and as glucose. He has arranged in ascending order: legumes, dairy products, fruit, biscuits, cereals, vegetables and sugars. (Jenkins, 1981b). He suggests that the lack of a significant relationship between the glycaemic index and dietary fibre content of wheat products, may be due to the fact that wheat fibre has little effect on blood glucose, as was mentioned earlier. The priming effect of high-CHO diets may have

accounted for the fact that cereal fibre has been shown occasionally to be effective.

Jenkins has coined the term "Lente Carbohydrate" to illustrate the importance of slow-release dietary carbohydrate on the blood glucose response (Jenkins, 1982). The glycaemic index, which is lowest for beans, lentils and legumes in general, may thus relate possibly to other components of foods besides fibre which alter digestibility, e.g. enzyme inhibitors, lectins, tannins, phytates, sugars, fats, proteins, starches, as well as the actual structure or form of the food. Legumes are rich in both fibre and many of these "anti-nutrients" – heating lentils for several hours diminished their low glycaemic index (Jenkins, Thorne, Camelon et al, 1982b), probably by destroying some of these factors.

Innovative as Jenkins' work undoubtedly is, his results are, however, clouded by the fact that the tests were done in an isolated rather than a meal situation. Ingestion of protein and fat with CHO has been shown to affect the elevation of blood glucose (Estrich, Ravnick, Schlierf et al, 1967). Also his diabetic subjects involved in the tests took their usual hypoglycaemic agents before ingesting the foods. Some patients even changed their dosage during the course of the study. On some occasions milk was taken with certain foods (e.g. breakfast cereals) but not with others, or tomato was added to cooked grains, legumes or pasta in order to increase palatability (Jenkins, Wolever, Jenkins et al, 1983b). These factors could all influence the final verdict on whether a certain food has a lower glycaemic index relative to another. However, one can assume that the general trend of his results is correct, i.e. that legumes in general have a smaller effect on post-prandial blood glucose than other classes of foods. They have also been shown to improve the glucose tolerance to a subsequent meal 4 hours later (Jenkins, Wolever, Taylor et al, 1982c) in a similar way to guar gum (Jenkins, Wolever, Nineham et al, 1980a).

A recent attempt to quantify the effect of different forms of CHO taken in meals, was made by Bantle, Laine, Castle et al (1983). Glucose, fructose, sucrose, potato and wheat starch were each tested in breakfasts with practically identical amounts of protein, fat and CHO. 49% of calories came from CHO in each meal. It was found that there was no significant difference in the time taken by subjects (healthy, IDDM and

NIDDM) groups) to reach peak blood glucose concentration with any meal type, and that the sucrose meals did not produce significantly greater peak increments of plasma glucose than other meals. It was thus concluded that dietary sucrose did not aggravate post-prandial hyperglycaemia when consumed as part of a meal. However, the fibre content of all meals was low, and the 42g of CHO given as rice cereal and milk may well have masked the effect of the various added test CHO.

Nevertheless, one can deduce that the new approach to diabetic meal planning must include more knowledge of the physiological effects of different carbohydrates taken in the meals. Thus the simple and time-honoured means of regulating diabetic diets by means of "CHO exchanges" based on chemical analysis of foods, may not predict the physiological response to the food. This response will depend on the nature of the starch, free sugar, fat, protein and fibre content, as well as the food form and the presence of inhibitors of enzymatic digestion. More work is clearly needed in this area, as foods which release their CHO slowly should be of advantage to diabetics. It is interesting to note that beans were used extensively as a source of high CHO high-fibre foods in the studies in Oxford. The enzyme inhibitors which they contain, are similar to that of Acarbose, a drug which has been tested recently for its effect on diabetic control. It would still seem prudent, on the weight of the evidence, to encourage a modified-fat, high CHO (50% dietary energy) diet for controlled non-obese diabetics, with restriction of mono- and disaccharides and encouragement of unrefined, high fibre foods, which release their CHO slowly in the gastro-intestinal tract.

Section B: Education of the diabetic patient

1. THE IMPORTANCE OF METABOLIC CONTROL

It has been calculated that diabetics are twice as prone to myocardial infarction, 17 times more prone to chronic renal failure, and 25 times more susceptible to blindness, than the general population (Crofford, 1976). Microvascular complications appear to be related to the degree of blood sugar control and the duration of the disease (Kahn and Klaff, 1983). Moreover, evidence is accumulating that excellent glycaemic control will delay or prevent the progression of these complications. In addition, it has been shown that in poorly controlled diabetics, a lipid pattern develops which is characteristic of increased risk of atherosclerosis, while the reverse is true with good control of blood sugar (Lopes-Virella, Wohltmann, Loadholt et al, 1981). Thus the therapeutic aim in the treatment of diabetes should be to achieve good glycaemic control, in an attempt to prevent or minimise the onset of complications.

Once we are committed to the view that control is beneficial, we need to assess the requirements of the patients in order to educate and train them to achieve these goals (Cahill, 1976).

2. ASSESSMENT OF THE PROBLEM

It has always been assumed that dietary adherence is the crux of diabetic control. It may well be an important prognostic risk factor for the development of complications (Hadden, Montgomery, Skelly et al, 1975). However, patients seldom follow their dietary recommendations (Williams, Anderson, Watkins et al, 1967; West, 1973). This was originally shown by Dahlberg (1947) in a survey of all known diabetics in Sweden during 1942-1943. Only 33,9% of males and 37,4% of females who replied to a questionnaire, reporting that they had been given dietary instructions, were in fact keeping to them strictly. 53% followed the diet less strictly and 10,9% were not adhering to the diet at all. The longer the patient had been using insulin, the more frequently he had changed to a "normal" free diet.

In a survey done in Leeds, (Tunbridge & Wetherill, 1970) it was found that dietary adherence was even worse. Here the food actually eaten by

more than ⅔ of the diabetic out-patients studied, was appreciably different from that prescribed. The diabetes supplement of the U.S. National Health Survey (Holland, 1968) showed that 25% of responding subjects did not follow their diet, while 22% indicated that they had never been given a diet.

On the other hand, dietary adherence has been shown to bear no relationship to the standard of diabetic control as recorded in the clinic. (Tunbridge & Wetherill, 1970). Other factors may therefore play a part. Poor control of diabetes seems to be associated with one or more of the following: Ignorance of the diabetic regimen, social or environmental difficulties, emotional problems or refusal of therapy (Stone, 1961).

The term "compliance" has been coined to describe the extent to which a person's behaviour coincides with the medical advice. (Haynes, Taylor and Sackett, 1979). The success of therapy with chronically ill, ambulatory patients will depend on their compliance to the therapeutic recommendations. Difficulties with compliance have been noted for centuries. Hippocrates remarked that the physician "should keep aware of the fact that patients often lie when they state that they have taken certain medicines". The rate of compliance to long-term medication is approximately 50%, and data indicate that the physician's estimate of patient compliance is of very limited value. Clinical studies have also failed to show a relationship between diabetic compliance and either age at onset, duration of diabetes, sex or other socio-demographic details (Etzwiler & Maiman, 1982).

One of the most important factors in achieving compliance seems to be the relationship between the patient and the physician (Tunbridge, 1953; Williams, 1967). This presumably acts by increasing the motivation of the patient. Patients who are less satisfied with their physicians are more likely to make errors in recalling their therapeutic regimen (Brody, 1980). The importance of the interaction of patient and therapist was shown in a study on drug compliance in ambulatory South African Black diabetic patients, where 20-30% of the patients studied, felt that the doctor's examination or explanation of the therapy was inadequate. Different socio-economic backgrounds of doctor and patient could also play a part (Buchanan, Shuenyane, Mashigo et al, 1979).

Patient recall of doctors' instructions may also be an important determinant of compliance, and it has been found this too depends on communication between doctor and patients (Hulka, 1976). Instructions should be clear and concise and the doctor should prescribe the minimum number of drugs consistent with the therapeutic goal, or focus on fewer items of instruction at each visit (Page, 1981).

The doctor should also assess the patient's beliefs concerning his disease. The "health belief model" (Ludvigsson, Richt and Svensson, 1980) argues that patients who view their illness as serious, do better clinically, provided that they have confidence that the medical treatment can help. It has been shown that a positive correlation exists between patients' overall compliance and their level of health belief (Cerkoney and Hart, 1980).

Whether or not an individual will undertake a recommended health action will thus depend on his perception of

(a) his personal susceptibility,
(b) the degree of severity of the consequences of the disease,
(c) the potential benefits of treatment, and
(d) the socio-economic barriers to the required behaviour.

It is more difficult to achieve adherence to therapeutic regimens which entail a change in lifestyle than those which simply require medication.

Compliance also depends on the personality of the patient. Generally speaking, patients may be divided into

(a) "internal" or
(b) "external" types (Lowery, 1976).

An internal type of patient is one who actively seks information pertaining to a situation, because he believes that a relationship exists between action and outcome. At the same time he may be more resistant to external attempts to influence his behaviour.

An external type, on the other hand, does not believe that efforts are connected with rewards, is more passive and will comply more fully with authority.

It has been shown that internal diabetic subjects obtain more knowledge about their disease, and know more about control-related activities in the

early stages after the diagnosis, but are not superior to external subjects in maintaining their health after a number of years. Thus the internal subject attempts to learn in order to control his disease, but if the information he obtains is not relevant, or not seen to be of benefit, then he will cease to comply, and may incur more problems.

The external subject will follow the prescribed regimen more fully over a long period of time, while he passively gains facts about his condition. The active seeking of information by the internal subject would be the preferred response, however, as long as he obtains relevant information which will aid him in achieving good control. Thus it is important to remember that patients vary in their response to the diagnosis of the disease, and to the instructions they receive. Externals do not require too much detail initially, and respond well to the health belief model, while internals, who are generally self-motivated, need a peer-partnership with the practitioner (Leichter & Chandler, 1982).

Patients who do not have confidence that medical treatment can help them, may exhibit more psychoneurotic complaints (Linn, Linn, Skyler et al, 1980) and may be more difficult to treat. This type of patient should, therefore, be targeted for therapy which would improve his psychological state.

In general, then, it seems that the achievement of diabetic control depends on multiple factors besides dietary adherence. Medical non-compliance may be a phenomenon or manifestation of poor adjustment or acceptance by the patient (Roberts & Wurtele, 1980) and may only be overcome with improved, more effective teaching and therapeutic tools.

3. EDUCATION OF THE DIABETIC PATIENT.

Patient education needs careful planning. One should first assess the patients and their needs (Kaufman, 1964), and then proceed with information geared to the patients' level of understanding and socio-economic status.

The ignorance of diabetics regarding their disease was highlighted by a survey done by Beaser (1956) in the New England area. He suggested stressing the permanency of the condition, a point which was also mentioned by Breidahl (1980). Many patients, particularly NIDDM,

might tend to think they were cured once their symptoms were alleviated, and thus might cease to follow instructions. Poor compliance could then produce symptoms of diabetic complications several years later. The patient should, therefore, be given a real understanding of the disease, and continual medical supervision.

Etzwiler (1972) has estimated that 6-12 hours need to be spent simply in dietary education. The diet needs to be tailored to the individual needs of the patient, and compliance requires modification of existing behaviour, as well as the acquisition of new behaviour, both in the dietary and medical areas of treatment.

The team approach to education is recommended, and the patient should be made to understand that he is part of the medical team, since chronic diseases require the participation of the patient in therapy. The educational programme should be planned in stages (Etzwiler, 1978) with basic information regarding the disease, treatment and procedures involved, being given in the initial stage.

Both group methods (Hassell and Medved, 1975) and one-to-one methods of instruction (Rabkin, Boyko, Wilson et al, 1983) have been shown to be effective. In group discussions anxiety may be reduced and peer reinforcement is obtained. The improvement in patient knowledge may persist for a short while (Chandalia & Bagrodia, 1979), and constant reassessment and reinforcement is necessary (Lawrence & Cheely, 1980).

The in-depth education could include further information on how to cope under adverse conditions, as well as motivation of the patient to carry out his responsibilities. Here it seems that blood-monitoring is an excellent means of reinforcing patient education regarding the effect of food, exercise and insulin adjustment (Peterson, Forham and Jones, 1980). The patient and his family should be taught a flexible regimen of insulin dosage (Skyler, Ellis, Skyler et al, 1979) to deal with variations in life-style and health.

The educator should realise how difficult total compliance is, and should not adopt a superior authoritative attitude. He should also not propound blind belief in the efficacy of treatment, as this would lead to disbelief and noncompliance after future problems. Similarly, hyperglycaemia should not always be assumed to be due to dietary

non-compliance (Sulway, Tupling, Webb et al, 1980), and the guilt for failure to achieve good glycaemic control should not be transferred solely to the patient. Joint management of the problem should be attempted.

Behaviour modification has a place in the educational process, (Surwit, Scovern and Feinglos, 1982) and techniques such as contracting and specific assignments have shown promise. Initially only the necessary basic demands should be made on the patient, even at the risk of some degree of blood glucose control, and gradually more behaviour changes can be required. Many objectives may only be achieved long after the initial diagnosis.

The importance of patient education is now recognised as an integral part of total health care. It is believed that the correct approach to patient education may help to achieve good diabetic control. However, several clinical studies have failed to find a relationship between improved knowledge and glycaemic control (Stone, 1961; Etzwiler, 1978). In fact an inverse correlation has been shown (Williams, 1967). Thus instruction is not enough – the patients must be motivated for compliance. Sustained behavioural changes require much communication and support. A tendency to improved control has been found in regular attenders at a clinic, despite an overall lack of knowledge in diabetic patients (Beggan, Cregan and Drury, 1982).

The goal of patient education then, should be not simply an increase in knowledge but improved compliance, in order ultimately to achieve better glycaemic control.

Chapter 2

AIMS AND OBJECTIVES

INTRODUCTION

It should be borne in mind that, at the time when these studies were designed, some of the data reviewed in the previous chapter was unpublished. Thus the aims and objectives described here, pertain to aspects of diabetic control which were under investigation at that stage, and so reflect concepts of diabetic therapy which were then relevant. They also reflect some issues which still remain uncertain in 1983.

The studies to be presented in this thesis are considered in three sections, each of which deals with a separate aspect of diabetic therapy. These are:

(1) The effect of a mass education programme on diabetic knowledge, compliance and control.
(2) The effect of a long-term high-fibre diet on the glycaemic control of diabetic out-patients.
(3) The effect of a supplement of viscous fibre on the control of diabetes in the out-patient situation.

THE RATIONALE FOR THESE STUDIES

(1) *Mass-education programme*

As described in the literature, assessment of the state of knowledge of the patients to be educated is essential before planning any educational programme. One must identify the problem (lack of knowledge in specific areas) and the type of patients who need the education, in order to design an effective educational tool. At the time that this project was planned, we had no current details of either of these aspects. Consequently this project was planned in three stages:

(a) To assess the level of existing knowledge, education and standard of glycaemic control in our diabetic clinic population, at the Outpatient Department of Groote Schuur Hospital.
(b) To use the specific educational deficits thus highlighted, as the basis

for constructing an effective educational programme, which would be shown repetitively to the clinic population until adequate exposure had been achieved.

(c) To re-assess the knowledge and education of the diabetic out-patients, after exposure to the educational programme, in order to ascertain whether the programme had in fact improved the level of knowledge in our patients, and whether this increased knowledge would lead to greater compliance with therapy and to improved glycaemic control.

Reports in the literature have indicated that diabetic control need not necessarily improve with improved knowledge of diabetes, but that ignorance of the diabetic regimen is associated with poor control. While we could not aim to improve the socio-economic, environmental or emotional problems which might also contribute to poor control, we hoped at least to remove the factor of ignorance about diabetes. We would then investigate whether increased knowledge would lead to greater compliance with therapeutic procedures and consequently to improved diabetic control. We would also be able to ascertain where the problem areas regarding compliance lay – with compliance to medication, to self-care techniques or to dietary recommendations.

(2) *Long-term high-fibre study*

This study was designed to ascertain whether the use of high-fibre diets was practical in the South African situation. Reports had been received of the work done by Anderson and Simpson on HCF diets, and their effect in improving the diabetic control of the outpatients studied.

Our aim was two-fold:

Firstly, to investigate the effect of locally-obtainable, low-cost foods rich in dietary fibre on the glycaemic control of our diabetic out-patients, and Secondly, to test the practicability of such a dietary regime for our typically poorly-educated patients of low socio-economic status.

The level of dietary intervention which is required to achieve an improvement in diabetic control, is such that it entails a drastic change in dietary habits. It was felt that such a diet might only succeed with well-motivated, well-educated patients. Anderson in fact has described his techniques for educating and motivating patients, commencing with a

seven-day period of hospitalisation in order to acquaint the patient with the required modifications. This is clearly not practical for all diabetic outpatients in this country.

We made no provision for such educational intervention, in order to evaluate the feasibility of compliance to dietary changes without introducing any other variables. Our objective was thus purely to assess the practicality of HCF diets in the local situation, and the efficacy of this as a tool to achieve better glycaemic control.

(3) *The use of guar gum as a supplement in the diabetic diet.*
Concurrently with the above 2 studies, we were also investigating the development of a palatable vehicle for incorporation of guar gum into the diabetic diet, and the efficacy of such a supplement in improving diabetic control. It had been demonstrated by Jenkins that guar gum was the most effective fibre which could be used as an additive to meals in order to produce better glycaemic control. However Jenkins had only used the gum in large doses (14-26g per day) and problems had been experienced with side-effects such as flatulence and abdominal distention in using these quantities, as well as with palatability of the viscous gum when added to foods. We, therefore, attempted to develop, with the help of a local biscuit manufacturer, a palatable vehicle for a small dose of guar gum, which could be taken regularly as a supplement to meals, and then tested the effect of this supplement in improving tolerance to a carbohydrate load in the short- and long-term situation. There would obviously be an advantage in using smaller doses of guar gum, in that the food supplement would be more palatable and one would be more likely to achieve long-term compliance. The biscuit, or vehicle for the addition of guar to the meal plan, would of necessity be high in carbohydrate, since guar has been shown to be most effective when intimately mixed with the carbohydrate of the meal, rather than when sprinkled onto the food.

The aim of this study, therefore, was to ascertain whether a small dose of guar given in a palatable form as a biscuit, was effective in improving carbohydrate tolerance, and whether it could be incorporated into the diabetic meal plan on a regular, long-term basis, in order to improve

glycaemic control. In order to achieve this situation, patients would have to comply with instructions on dietary modifications which would be necessary to incorporate the biscuits into their meal plans.

SUMMARY

The ultimate objective in all these studies was thus to evaluate different methods of improving diabetic control. The methods chosen for investigation were: improving diabetic knowledge, increasing the intake of high-fibre foods, and the use of a supplement of viscous fibre. The success of each of these methods would depend on compliance of the ambulant diabetic patient with instructions received. This study is, therefore, a practical one which aims to assess the ease of implementing each method of improving diabetic control, an issue which has become important as evidence has mounted for the correlation between poor glycaemic control and the incidence of diabetic complications.

Chapter 3

THE EDUCATION PROJECT

INTRODUCTION

The aims of this study have been described in detail in Chapter 2. Essentially it was hoped that this study would firstly uncover specific educational deficits in the out-patients attending the Diabetes Clinic, and also provide information which could be used to construct an effective educational programme, thus improving all aspects of diabetic care. Statistical comparison of the results obtained by testing a random sample of patients before and after the educational programme, would indicate any change in knowledge, education, compliance and diabetic control in the clinic population.

METHODS

(a) *The questionnaire*

A detailed questionnaire was drawn up, a specimen of which is shown in Enclosure I, to be used as the tool for assessing knowledge, education and compliance in our diabetic patients. This was divided into 5 broad sections, namely:

(1) Demographic data to be used for statistical analysis
(2) General diabetic information
(3) Diabetic dietary information
(4) Specific questions for NIDDM
(5) Specific questions for IDDM.

Each section, apart from the demographic data, was sub-divided into questions pertaining to existing knowledge, education (concerning diabetes) which had been received prior to the questionnaire, and compliance with instructions received. A score was assigned to each question, and total scores for knowledge, education and compliance were thus obtained for sections 2-5.

(b) *Details of the scoring system*

Section 1 consisted of information related to the patient, i.e. age, sex,

race, type of diabetes, weight, duration of diabetes, number of hospital admissions, form of therapy, standard of diabetic control, glycosylated haemoglobin, doctors' opinion of compliance to diet and medication, and level of education of the patient. Scores here were coded directly for computer analysis.

Section 2 assessed the patients "general knowledge" of diabetes, and begins with question 4 on the second page. The questions covered the patients' concept of the nature of the disease, urine testing and foot care. Questions 2, 3 and 17 were only included in the questionnaire used to retest the patients after the education programme, and not in the initial assessment. Questions 4 to 11 tested the patients' "knowledge", questions 12 and 13 assessed the "education" in this respect (i.e. what the patient had been told), and questions 15 and 16 assessed the patients' "compliance".

Section 3 covered all aspects of the diabetic diet in great detail. Here questions 1-9 related to "knowledge", questions 10-21 related to "education", and questions 22-29 related to "compliance" in this section.

Section 4 was only completed by patients on oral anti-diabetic agents, and tested these patients' knowledge of their drugs with questions 1-3, and drug compliance with questions 4 and 5.

Section 5 was for patients on insulin therapy, and tested their understanding of the actions and use of insulin, as well as injection technique and compliance. Here question 1 was only included in the second questionnaire, i.e. after the education programme. Questions 2-14 assessed knowledge, and questions 15-19 assessed technique and compliance.

In sections 2-5 a simple binary system was used for scoring, e.g.

Yes = 1, No = 0

Pass = 1, Fail = 0

Where questions required more than one answer, e.g. question 4 of section 2, or question 5 of section 3, a certain minimum number of "ticks" was required to obtain a pass, or a score of 1, and a half-mark was deducted from the total correct number for each error made.

The final scores for each subgroup, i.e. knowledge, education and

compliance, thus obtained in sections 2-5 of the questionnaire, were subjected to statistical analysis together with the coded demographic data from section 1. This was done in the Dept of Mathematical Statisticsd of the University of Cape Town.

(c) *Statistical Methods*

The basis for the statistical analysis was an investigation to see whether one variable (the dependent variable) was affected significantly by another (the independent variable). The independent variables selected were: Duration, Age, Sex, Race, Treatment, Education-level (which was taken to indicate socio-economic status), Doctor's opinion on dietary compliance, Doctor's opinion on medication compliance, and Education received.

The dependent variables were:

General knowledge, education and compliance
Dietary knowledge, education and compliance
Tablet knowledge and compliance
Insulin knowledge and compliance, and
Standard of control.

Analysis of the data was carried out in the Dept of Mathematical Statistics at UCT on a Univac 1100 computer. Different statistical tests were used according to whether the variables measured were interval (e.g. diabetic control), ordinal (e.g. age or knowledge intervals), or nominal (e.g. education received). Where classification was ordinal, the Kendall's Tau-B statistic was used, and where classification was nominal, the contingency coefficient was used. The Chi-squared test was also used as a measure of association between the variables. Details of the statistical methods are given in the Appendix.

(d) *Completion of the questionnaire*

A team of 3 para-medical co-workers distributed the questionnaires to a random sample of patients in the waiting bays of the Diabetes Clinic in the Out-patients' Department of Groote Schuur Hospital. This was done both before and after the education programme. Certain patients requried help in completing the questionnaires, either because of poor

eyesight, illiteracy or lack of comprehension of this unfamiliar form of testing. Assistance was then given in such a way that no bias was introduced, and the co-workers were coached in methods of phrasing the questions or in the recording of the answers. If no help was necessary, patients were allowed to complete the questionnaires on their own, and the completed form was checked by one of the co-workers to ensure that no section or questions had been omitted.

(e) *Assessment of Diabetic Control*

Capillary whole blood glucose is routinely measured in diabetic out-patients with a Beckman Glucose Analyser immediately before each clinic visit. The mean of the previous year's blood glucose values was calculated, and on this basis each patient who completed a questionnaire was assigned into one of the following categories:

Diabetic Control	*Mean Blood Glucose*
Excellent	**≤ 8.5 mMol/1**
Average	**8.6-11.0 mMol/1**
Fair	**11.1-13.9 mMol/1**
Poor	**≥ 14.0 mMol/1**

Glycosylated haemoglobin (HbA_1) was assayed in 25% of the patients in each sample group, i.e. before and after the education programme, using the short-column ion-exchange chromatographic method. (Kynoch, 1977).

(f) *The Educational Programme*

Based on the educational deficits which became apparent on analysis of the scores obtained in the initial part of this study, an educational programme was devised. This consisted firstly of a number of impulsed slide-tape programmes, which were recorded in the Audio-visual Education Unit of the College of Medicine of S.A., based in Rondebosch, Cape. The scripts and art-work for the slides were devised in the Department of Medicine, by the author and other members of the Diabetes and Endocrine Research Group. The contents covered the physiology of diabetes, the therapeutic agents, methods of self-care and self-assessment of diabetic control, and dietary instruction.

In addition to the slide-tape programmes, which enabled group

education (under the supervision of the author) to take place, a diabetes education sister was employed to provide individual counselling for patients with specific problems. We also introduced record cards, which were attached to patients' appointment cards, to provide them with a personal record of their progress (or otherwise) as regarded weight and blood sugar at each visit ot the clinic. Printed notes were also supplied to all patients in the waiting bays, summarising the main points of self-management for IDDM and NIDDM. Copies of these notes are enclosed (Enclosure II).

Our educational sessions had to be tailored to the organisation of the clinic, since for socio-economic reasons patients generally were not able to attend the hospital on a separate occasion solely for the purpose of education. The system in the Diabetes Clinic is such that patients arrive at approximately 08h00, queue to have their blood glucose estimations and then wait in the passages or in the waiting bays until they are called to the doctor. We therefore used an informal approach, by requesting groups of patients to sit in the waiting bay designated for our use, and to view the programmes while they waited to be called into the clinic.

Thus, it was not possible to document the length of exposure of each patient to the education programme, nor was it possible to isolate the patients who were being educated, from a control group who would receive no education. Neither was it possible to test two identical samples, because of the difficulty in tracing patients and recalling them for special education. The random method used, was however thought to be more relevant to the situation in our clinic than a more controlled, but artificially precise method such as used overseas.

(g) *Summary of methodology*

We thus tested, with the same questionnaire and the same scoring system, a random sample of clinic patients in order to assess the existing knowledge in the clinic prior to commencing the education programme, and another random sample of patients who had viewed the audio-visual programmes, after a suitable period of exposure to the slide-tape programmes. In this way, with statistical analysis of the

scores before and after the programmes we attempted to assess the effect of these programmes on knowledge, education and compliance in our diabetic out-patient clinic as a whole, and not in individual patients. We also investigated whether any increase in knowledge that might be produced, would lead to improved diabetic control.

RESULTS OF THE TWO SURVEYS

(a) *Demographic data*

Figure 3.1 represents the relevant demographic data of our 2 samples.

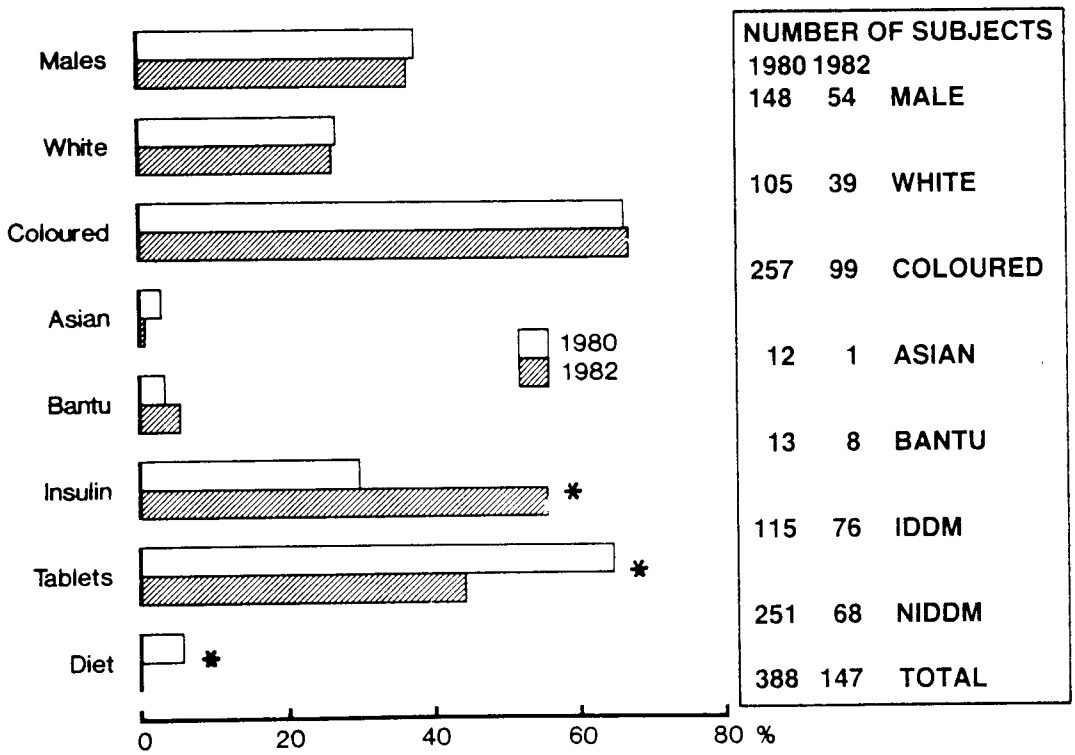

Fig. 3.1 Bar diagram showing demographic data of the two samples
*** $p < 0.01$**

It can be seen that there was no statistical difference between the sex and race distribution of the 2 samples. The mean age of the 2 sample groups was approximately equivalent, as shown below:

Year	*Mean Age (years)* (±SD)
1980	51.7 ± 14.7
1982	48.5 ± 14.3

There was also no significant difference in the educational levels, which were used as an index of socio-economic status, as shown by the percentage falling into each interval or category in Table 3.1 below:

Interval	*1. Illiterate*	*2. Lower Primary*	*3. Higher Primary*	*4. High School*	*5. Matric*	*6. Technical*	*7. University*
1980	3.6	20.1	31.8	31.2	7.6	2.1	3.6
1982	6.1	9.5	29.9	36.1	10.2	2.0	6.1

TABLE 3.1 Distribution of patient education levels in the two samples.

On statistical analysis the mean was given as 3.4 (±1.4) intervals in 1980, and 3.7 (±1.4) intervals in 1982. Thus it fell between Higher Primary (interval 3) and High School (interval 4) in each sample group. The Chi-squared statistic for the comparison of these two groups was calculated to be 11.65, with a p value of <0.1. Thus this also shows the lack of statistical difference between the two samples.

The appearance of the patients in our two samples was also statistically similar, as shown by the percentages falling into each category or interval in Table 3.2 below:

Interval	*1. Under-weight*	*2. Normal*	*3. Over-weight*	*4. Obese*	*Mean interval (±SD)*
1980	4.2	51.6	30.7	13.5	2.5 (±0.8)
1982	2.7	58.5	32.6	6.1	2.4 (±0.7)

TABLE 3.2 Distribution of patient appearance in the two samples.

The Chi-squared statistic here was calculated to be 6.74, and the *p* value <0.1, i.e. there was no statistical difference between the two samples in this respect.

The distribution of the duration of diabetes was also similar in the two samples, as shown by the percentages of subjects falling into each category or interval in Table 3.3 below:

Interval	*1. <1 year*	*2. 1-3 yrs*	*3. 3-5 yrs*	*4. 5-10 yrs*	*5. >10 yrs*	*Mean interval (±SD)*
1980	6.5	15.1	15.4	27.6	35.4	3.7 (±1.3)
1982	4.8	13.6	8.2	26.5	46.9	3.9 (±1.2)

TABLE 3.3 Distribution of duration of diabetes in the two samples.

The Chi-squared statistic here was equal to 8.53, with a p value of <0.1, i.e. again no statistical difference between the samples.

We were thus satisfied that the two samples were comparable statistically. However from Figure 3.1 it can be seen that there was a very highly significant difference in the distribution of the form of treatment of the patients tested in the two samples, there being more insulin users in the second sample. It must be remembered that our second sample was selected from patients who had viewed the films. Thus insulin-users had shown more interest in viewing the films and in learning about their disease than NIDDM, when invited to sit in the waiting bay for the education programme.

Because of the difference in distribution of the treatment of the patients in the two samples, we first compared the results of the education programme in groups of insulin-users and non-insulin users separately.

(b) *Analysis of scores*

(i) *Insulin-users:* Here the total scores for general knowledge (GKN), general education (GED), general compliance (GCOMP), dietary knowledge (DKN) and dietary education (DED) all improved very significantly in the sample tested after the education programme, as shown by the p values of the Mann-Whitney U-test for comparison of two independent samples. However, dietary compliance scores (DCOMP) showed no improvement, as indicated in Table 3.4 below.

Variable	*p value*
G KN	<0.0001 – very highly significant.
G ED	<0.0001 – very highly significant.
G COMP	<0.001 – very highly significant.
D KN	<0.0001 – very highly significant.
D ED	<0.0001 – very highly significant.
D COMP	0.18 – not significant.

TABLE 3.4 Significance of improvement in scores of IDDM

(ii) *Non-insulin users:*

Here again, the total scores for all the sections except dietary compliance, improved significantly after the education programme, as shown in Table 3.5.

Variable	*p value*
G KN	<0.0001 – very highly significant.
G ED	<0.0001 – very highly significant.
G COMP	<0.0001 – very highly significant.
D KN	<0.0001 – very highly significant.
D ED	<0.0001 – very highly significant.
D COMP	0.36 – not significant.

TABLE 3.5 Significance of improvement in scores of NIDDM

Thus we found the same pattern of improvement in knowledge, education and compliance, excepting dietary compliance, in both insulin- and non-insulin-users. We therefore combined the data in order to compare the mean scores for all patients before and after the education programme. Here again all scores improved after the education programme, except dietary compliance, while the most marked improvements occurred in general knowledge and dietary knowledge. This is illustrated in the bar diagram below.

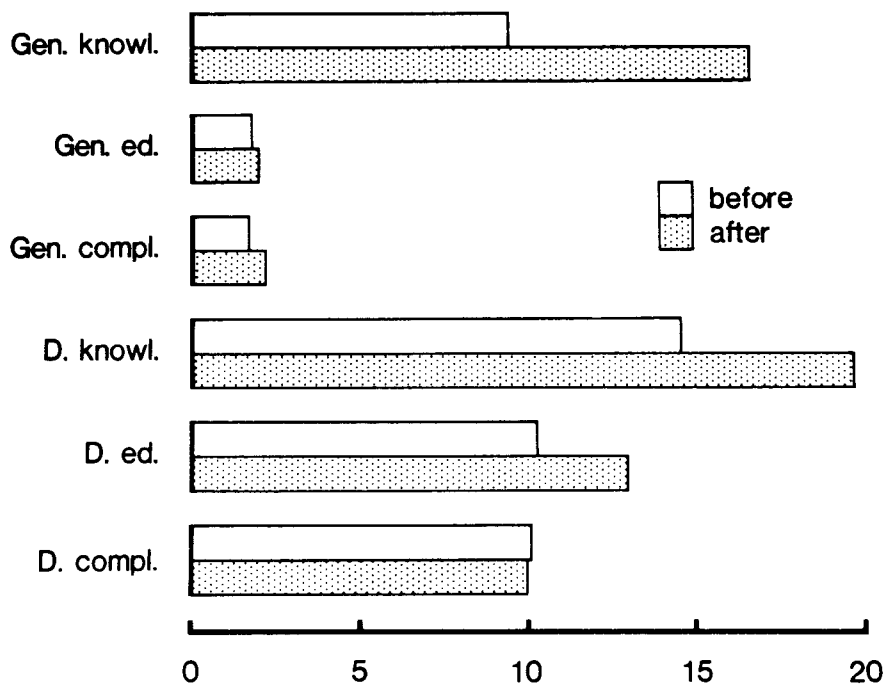

FIG. 3.2 Bar diagram showing mean scores in the two samples.

(c) *Comparison of diabetic control*

This parameter wass assessed in two ways, as has been described in the methodology:

(i) in categories or intervals of excellent, average, fair and poor, based

on the mean of the previous year's blood glucose estimations on routine clinic visits, and

(ii) by assessment of HbA_1 in approximately 25% of the patient sample tested by questionnaires.

Despite the improvement in knowledge which was produced by the education programme, no improvement in the standard of diabetic control was found by either of the above methods of assessment, as shown in Table 3.6.

Year	*Mean standard of control*	*Interpretation*	*HbA_1 (mean ±SD)*
1980	Mean interval 2.6	Average to fair	9.01 ± 2.7
1982	Mean interval 2.5	Average to fair	10.62 ± 3.4

TABLE 3.6 Comparison of diabetic control.

The Chi-squared statistic for the comparison of the standard of control in the two samples, was calculated to be 8,25, with a *p* value of <0.1.

(d) *Analysis of the Variables*

(i) *Doctors' opinion of compliance.*

When the doctors' assessment of compliance with medication (MEDCOMP) and diet (DCOMP) was compared with the actual compliance scores of the patients, by Chi-squared contingency tests, we found that in both sample groups, the doctors were only assessing the patients' medication compliance accurately, while their opinion of the patients' dietary compliance correlated less well with the actual compliance in this respect. The probability values for the comparison of the doctors' opinion with the actual compliance, are given in Table 3.7 below.

Year	*MEDCOMP*	*DCOMP*
1980	0.04	0.08
1982	0.03	0.08

TABLE 3.7 p values for correlation of doctors' opinion of compliance with actual compliance.

(ii) *Correlation of age of patient with other variables.*

There was a negative correlation of general knowledge, general education and general compliance with age, in both sample groups, as is shown in the following table by the Kendall's Tau-B correlation coefficient (with standard deviations).

Year	*Age vs G KN*	*Age vs G ED*	*Age vs G COMP*
1980	–0.28 (± 0.03)	–0.27 (± 0.04)	–0.29 (± 0.04)
1982	–0.26 (± 0.07)	–0.20 (± 0.07)	–0.24 (± 0.07)

TABLE 3.8 Correlation of age with knowledge, education and compliance.

(iii) *Association of control of diabetes with other variables.*
The standard of control, as assessed by the mean of the previous year's blood glucose estimations, was not associated with either general knowledge of diabetes, general education or general compliance, as shown in the following table, which gives the Kendall's Tau-B coefficients and standard deviations.

Year	*Control vs G KN*	*Control vs G ED*	*Control vs G COMP*
1980	–0.09 (± 0.04)	–0.03 (± 0.05)	–0.09 (± 0.04)
1982	–0.04 (± 0.07)	–0.03 (± 0.08)	–0.02 (± 0.08)

TABLE 3.9 Correlation of diabetic control with knowledge, education and compliance.

We also found that the standard of control of diabetes was not significantly associated with age in either sample, as shown by the Kendall's Tau-B coefficient and standard deviations, in Table 3.10.

Year	*Statistic*
1980	–0.04 (± 0.04)
1982	–0.09 (± 0.07)

TABLE 3.10 Correlation of control of diabetes with age.

(iv) *Association of form of treatment with other variables.*
Patients on insulin therapy scored significantly better in knowledge, education and compliance after the education programmes than other patients. The Kendall's Tau-B statistic and standard deviations are given below:

G KN	*G ED*	*G COMP*
0.26 (± 0.08)	0.34 (± 0.07)	0.26 (± 0.07)

TABLE 3.11 Comparison of improvement in scores with form of treatment.

The form of treatment was also found to be significantly related to the standard of control in our second sample (contingency coefficient = 0.27), thus showing that the insulin users were better controlled than those patients on oral agents.

(v) *Effect of education through the sister.*
The knowledge of insulin technique in those patients who received individual instruction from the diabetes education sister, was significantly associated with the education they received (contingency coefficient = 0.31). However, their scores on insulin compliance were not associated with this special education (contingency coefficient = 0.07), nor was this education associated with compliance to medication (contingency coefficient = 0.02). Education through the sister was also positively correlated with the general knowledge, education and compliance of the patients seen by her (contingency coefficients 0.15, 0.32 and 0.38 respectively).

(vi) *Effect of education through the films.*
The audio-visual method of education was most significantly associated with knowledge concerning insulin technique (contingency coefficient = 0.46) and insulin compliance (contingency coefficient = 0.27). The viewing of the films was also correlated with scores of general knowledge, general education and general compliance, as shown by the contingency coefficients of 0.25, 0.28 and 0.22 respectively.

DISCUSSION

It thus appears from the above data that the education programme was associated with a marked improvement both in knowledge pertaining to diabetes in general, and in dietary knowlege, as well as a slight improvement in general compliance to instructions regarding self-care. The increased knowledge was found to be significantly related to the intervention, i.e. the education programme, by the statistical correlation which was found between the viewing of the films and the variables analysed as shown in the results reported above. Individual education throught the sister was shown to be particularly associated with increased insulin knowledge, while general knowledge was less significantly associated with her education than with the viewing of the audio-visual

programmes. Thus the lack of a control group would not appear to hinder our deductions regarding the effectiveness of the education programme.

Despite the increased knowledge, however, dietary compliance did not improve, and in fact the mean scores remained essentially the same. This may have been a contributing factor to the lack of improvement in diabetic control demonstrated on analysis of the two samples, both in "standard of control" and in mean HbA_1 readings. Nevertheless, one should bear in mind that these indices have their limitations – the use of the mean of single blood glucose values obtained over a year on clinic visits may not be a good index of improvement or of assessment, as some of the figures used to obtain the mean, may have represented blood sugars for a period before exposure to the education programme, or before effective medical therapy was achieved. Similarly, the glycosylated haemoglobin figures may not have been truly representative of the clinic population as a whole. However, our results are similar to those reported in the literature where improved knowledge did not lead to improved diabetic control.

One should also remember that dietary compliance itself need not be the sole reason for poor control of diabetes. As has been shown in the literature, a multiplicity of factors interacts in the association of knowledge, compliance and control, amongst these one of the most important being communication between doctor and patient. It was shown that this clearly still needs to improve, since the doctors could only accurately assess their patients' compliance with instructions concerning medication, and not with those concerning diet. Also, as education should have as its basis a team approach, the dietitian should have closer liaison with the doctor regarding the patients' compliance and any factors which may affect this.

Similarly, the doctor should not transfer all the responsibility for improvement in glycaemic control on to the patient, with an exhortation to "stick to your diet". He should be aware that other factors could be affecting lack of compliance and poor glycaemic control, in particular imperfections in the existing therapeutic modalities for diabetes. The patient with an internal locus of control, as discussed in the literature, may become less compliant as he discovers that the information which he receives about his disease and the therapy which he requires, does not

appear to be effective in achieving good control.

The lack of association of knowledge with standard of diabetic control is interesting. The statistical analysis here supports our findings that, while knowledge scores increased after the education programme, glycaemic control did not improve. Thus this method of attempting to improve diabetic blood glucose levels, by means of education, does not appear to be very successful.

It is nevertheless gratifying to find that the education programme which we designed, has apparently been effective in increasing the knowledge of our diabetic out-patients. Perhaps, given more time, this improvement in knowledge could produce a more marked effect on the glycaemic control in the clinic population. We do feel that we have improved the awareness of diabetes in the clinic, and patients have responded favourably to the increased interest shown in their disease. This may have the effect of improving patient satisfaction with their therapy, and thus may indirectly influence compliance.

RECOMMENDATIONS FOR THE FUTURE.

As far as the situation in the Diabetes Clinic at Groote Schuur Hospital is concerned, it should be useful to record all patient data in a computer base, and then use this information to regroup the patients into clinic groups which have more homogeneous educational levels and therapeutic requirements. If this could be done, then the education programme could be expanded and aimed at specific target groups, in order to increase motivation, group responsiveness and possibly behaviour modification. This would hopefully have the effect of increasing compliance as well as knowledge of specific self-care regimens, for example adjustment of insulin dose and blood monitoring techniques for the relevant groups. In this way diabetic control may be improved in the important target groups, i.e. the young, insulin-dependent, motivated patients. It would also be useful to analyse the hospital statistics on the number of admissions for acute episodes of poor control of diabetes, in order to see whether these have decreased since the education programme was introduced.

Since insulin-users appear to score better on knowledge about diabetes, and since a significant negative correlation has been shown between

duration of the disease and control, as well as between the patient age and knowledge scores, future educational programmes should also be aimed at the older patients or those who have had diabetes for an appreciable length of time, and especially at the NIDDM patients. Thus each of the various categories and groups of patients could be educated in the clinic, with detailed information which would be related specifically to their requirements.

Other educational methods should also aim at improving dietary compliance, perhaps by assessing the reasons for non-compliance and seeking to rectify them. Possibly individual counselling here may have a greater effect in motivating patients to comply with their dietary guidelines, as meal plans could then be tailored to suit each patient's particular socio-economic needs and food preferences.

CONCLUSION

It therefore appears that our educational programme was associated with an improvement in knowledge of diabetes, but that increased knowledge did not correlate with improved compliance or diabetic control. Compliance both to dietary and therapeutic recommendations is difficult to achieve. Control of diabetes is probably affected by the combined action of many factors, not all of which have perhaps been analysed in this study. We can observe from this study the extreme difficulty which is encountered when attempting to improve diabetic control by educational means, particularly in the clinic situation.

ADDENDUM

Design of the study

We acknowledge that the second group of patients were to some extent "auto-selected," in that they were a random sample of those patients who had chosen to sit in the relevant waiting bay and thus exposed themselves to the education programme. Analysis of the distribution of form of treatment did in fact reveal that more insulin-users had availed themselves of the education programme. We deduced from this that insulin-users had shown more interest in the need for self-education, and allowed for this by analysing the scores obtained for insulin- and non-insulin-users separately. The same pattern of improvement in scores was obtained for both groups. Thus we felt justified in comparing the total scores obtained in each sample group.

Chapter 4

THE HIGH-FIBRE DIETARY STUDY

INTRODUCTION:

At the time when this study was planned, reports had been received of the efficacy of unabsorbable carbohydrates, when added to the meals of diabetic patients, in reducing the rise of post-prandial blood glucose levels in the short-term situation, and in improving diabetic control in the long-term. Because the gel fibres, guar and pectin, were reported to be somewhat unpalatable, and because all the long-term studies described in the literature had thus far been attempted in countries outside South Africa, we desired to incorporate readily-available local high-fibre foodstuffs into the diets of patients representative of our local out-patient diabetic population. We would thus attempt to ascertain the feasibility of changing the dietary habits of our typical, poorly-educated patients, who were of low socio-economic status, and also to investigate the effect of such high-fibre foods on the long-term glycaemic control of these patients.

METHODS

(a) *Patients and Study Protocol*

Eighteen patients from the Diabetes Clinic at Groote Schuur Hospital volunteered to take part in this study. Details are given in Table 4.1.

TABLE 4.1 PATIENT DETAILS

Patient	Sex	Age	Duration of DM (yrs)	Therapy	% Average body wt*
1	M	54	5	Diet	103,7
2	M	65	20	Oral	87,7
3	F	46	4	Oral	153,3
4	F	52	8	Oral	94,4
5	F	46	1	Oral	113,1
6	F	22	3	Insulin	93,5
7	M	62	10	Oral	89,5
8	F	50	3	Oral	117,4
9	M	59	5	Oral	111,0
10	M	53	10	Oral	74,7
11	F	57	10	Oral	104,2
12	F	52	10	Insulin	77,5
13	M	68	19	Insulin	75,0
14	M	38	4	Insulin	73,1
15	F	42	10	Oral	93,4
16	F	31	6	Insulin	116,4
17	F	51	2	Oral	101,6
18	M	41	4	Oral	98,9

* Geigy *Scientific Tables*, 7th ed., 1970.
DM = diabetes mellitus.

Unfortunately only 10 patients were able to complete this long-term study, because of various social, medical or psychological reasons. The study was divided into three periods, each lasting 12 weeks. During the entire duration of the study, patients were visited at two-week intervals, regularly at the same time of day, either at home or at work, where they were weighed. Venous blood was taken at each visit for assay of glucose, glycosylated haemoglobin (HbA_1), serum triglycerides, total cholesterol and high density lipoprotein (HDL) cholesterol.

The first 12-week period, termed the "uncontrolled diet" period, involved no intervention, and patients were kept on their usual diets for the entire period. This period was incorporated into the study in order to exclude a possible placebo effect of frequent visits by the doctor and dietitian on the patients' compliance and dietary adherence.

The second 12-week period involved "correction" of the patients' usual diet to a standard low-fibre diabetic diet, as was then in use for in-patients and out-patients of Groote Schuur Hospital. This period was termed the "low-fibre" period. During the third 12-week period, the patients' low-fibre diets were modified to incorporate increased amounts of dietary fibre, using foods which had been donated by various local manufacturers. This period was termed the "high-fibre" period.

The patients' anti-diabetic medications were not altered during this study, except for patient 14, who required a reduction in insulin dosage during the high-fibre diet period because of hypoglycaemic episodes.

(b) *Laboratory methods.*

Blood for glucose and HbA_1 estimations was collected in a fluoride tube, and plasma glucose was measured by the glucose oxidase method, on a Technicon autoanalyser. HbA_1 levels were estimated using short-column ion-exchange chromatography.

Serum triglycerides were measured using the fully-enzymatic UV method (Boehringer Mannheim kit) on the Technicon Autoanalyser, and total and HDL cholesterol levels were measured spectrophotometrically, using the Liebermann-Buchard reagent.

(c) *Statistical methods.*
Results of the laboratory assays were analysed using the Student's t – test for unpaired samples.

(d) *Dietary methods*
A master copy of the weights of portion sizes and the composition of the various foods typically eaten was drawn up using the dietary fibre and food composition figures published by Paul & Southgate (1978). Additional information was provided by the dietitians of the National Research Institute for Nutritional Diseases at Tygerberg, Cape. Each patient on the study was instructed in detail on how to keep a record of his daily intake of all food and drink consumed. A seven-day food record was obtained from each patient during the second 4 weeks of each 12-week period, and these food records were analysed manually with the use of a calculator and the master copy of food composition, in order to determine the average daily intake of energy, protein, fat, carbohydrate and dietary fibre for each patient during each 12-week period. A dietary analysis work sheet and examples of instructions to patients are included (Enclosure III).

During the uncontrolled period patients were kept on their own interpretation of the diabetic diet which had been given to them at the hospital. Information gained by the food records obtained during this period was used to devise an individualised low-fibre diabetic diet for the second period of the study, keeping to the patients' usual eating patterns. The carbohydrate content of the diet was calculated to be approximately 45%. The energy intake was tailored to the individual requirements, and calculated to be weight-maintaining.

During the third period these low-fibre diets were modified by including the high-fibre foodstuffs which had been donated. These were Weetbix cereal, digestive bran, dried prunes, canned baked beans and canned peas. Wholewheat bread was also substituted for white bread or other breads and biscuits which had been used in the low-fibre period. The amounts of each foodstuff were calculated for each patient so that the dietary fibre intake would be increased to 25g total dietary fibre per 4200 kJ per day. Exact instructions were given on the portion sizes of each food and the meal plan to be followed. The other carbohydrates and fats taken

in the habitual diet were adjusted so that the energy content of each patient's diet remained constant. A sample set of instructions for 2 of the patients is enclosed (Enclosure IV).

The percentage of dietary fibre (DF) in the foodstuffs used, is given in Table 4.2 below.

Type of food	% DF
Weetbix	**12,7**
Bran	**44,0**
Baked beans	**7,3**
Peas, canned	**6,3**
Prunes	**16,1**
Wholewheat bread	**8,5**

DF = dietary fibre

TABLE 4.2 Percentage of dietary fibre in foodstuffs used

The dietary fibre was predominantly of the non-cellulose polysaccharide type, and thus consisted chiefly of pectic substances and hemicelluloses.

On each visit the patients' compliance with the diet was checked and they were encouraged to adhere to their meal plans. The supply of the high-fibre foods to the patients at no cost, and the delivery to their homes of these foods, obviated any possible non-compliance for financial or other socio-economic reasons such as lack of transport to and from the shops.

RESULTS

The analysis of the food records kept by each patient who completed the study, during each of the three periods, is given in Table 4.3.

It will be seen from this table that, while all patients increased their dietary fibre intake during the high-fibre period, only 3 patients, numbers 11, 17 and 18, approached the projected fibre intake of 25g/4200 kJ per day, and not one attained it. The total intake of dietary fibre in each period by each patient is calculated in Table 4.4.

It will be seen that patients 4, 13 and 14 increased their dietary fibre intake from the uncontrolled to the low-fibre period. This was due to the correction of their diets to a standard 45% carbohydrate diet in the second period, which changed the composition of the diet from one of high protein, high fat, to higher carbohydrate, in accordance with the principles

TABLE 4.3 Food Intake

	Uncontrolled period					Low-fibre period					High-fibre period					
Patient	kJ	Prot. (g)	Fat (g)	CHO (g)	Fibre (g/4 200 kJ)	kJ	Prot. (g)	Fat (g)	CHO (g)	Fibre (g/4 200 kJ)	kJ	Prot. (g)	Fat (g)	CHO (g)	Fibre (g/4 200 kJ)	% incr
3	6 926	76,6	82,4	158,7	7,8	5 610	64,7	80,1	95,6	5,9	6 720	58,0	33,7	125,0	15,4	161,0
4	8 145	89,6	102,4	173,5	7,3	7 101	70,9	83,6	175,2	9,0	6 988	84,5	87,8	162,2	16,2	80,0
5	7 524	79,3	108,9	210,3	7,9	6 524	79,8	87,3	116,6	5,2	6 496	93,4	72,4	214,4	16,2	211,6
7	7 671	91,1	85,0	184,2	9,2	6 074	77,2	73,9	121,4	9,1	5 822	69,0	60,4	132,5	14,6	60,4
8	10 126	100,9	124,4	227,8	9,0	6 802	82,9	82,3	145,8	8,9	5 719	72,4	66,6	125,7	16,3	83,1
11	5 672	61,2	63,2	153,3	13,8	5 320	58,9	69,9	121,2	8,5	5 467	58,1	57,7	147,0	22,6	165,9
13	9 177	123,6	127,0	142,8	4,3	8 015	113,8	99,7	145,9	7,7	7 310	90,4	65,3	228,7	18,8	144,2
14	9 981	117,7	123,0	215,6	8,5	11 612	119,6	118,1	294,9	9,2	10 771	125,1	125,3	213,2	17,9	94,6
17	8 624	85,1	109,4	195,5	10,0	6 375	53,9	91,7	127,8	7,7	6 943	70,2	75,6	184,0	22,3	189,6
18	8 463	97,3	122,3	145,6	7,0	8 048	97,3	115,4	130,8	5,4	6 951	81,5	86,0	148,2	20,3	275,9

CHO carbohydrate

Patient	Uncontrolled period	Low-fibre period	High-fibre period
3	12.9	7.9	24.6
4	14.2	15.2	27.0
5	14.2	8.1	25.1
7	16.8	13.2	20.2
8	21.7	14.4	22.2
11	18.6	10.8	29.4
13	9.4	14.7	32.7
14	20.2	25.4	45.9
17	20.5	11.7	36.9
18	14.1	10.3	33.6

TABLE 4.4 Fibre Intake (g/day)

of the Groote Schuur Hospital diets. This step was deemed necessary in view of the evidence that a higher fibre intake would only be effective above a minimum of a 40% carbohydrate intake, as shown by Jenkins in the review of the literature.

The changes in body weight are shown in Table 4.5 below.

Patient	Uncontrolled period	Low-fibre period	High-fibre period
3	105.7 (± 1.5)	107.0 (± 1.2)	106.7 (± 1.4)
4	54.7 (± 0.3)	53.6 (± 0.5)*	54.1 (± 0.6)
5	68.7 (± 1.2)	68.9 (± 1.1)	67.9 (± 0.7)
7	57.0 (± 0.9)	56.0 (± 0.8)*	56.3 (± 0.6)
8	71.8 (± 1.0)	72.6 (± 0.5)	72.9 (± 0.7)
11	71.2 (± 0.9)	69.0 (± 1.9)*	70.1 (± 0.5)
13	65.9 (± 1.8)	64.7 (± 1.4)	63.2 (± 1.3)≠
14	59.3 (± 0.9)	60.1 (± 0.9)	60.3 (± 0.4)
17	71,9 (± 1.3)	70.3 (± 0.9)*	70.1 (± 0.6)
18	77.6 (± 0.8)	77.9 (± 0.7)	77.0 (± 0.9)

TABLE 4.5 Body weight (mean ±SD) in Kg

* indicates significant weight change from uncontrolled to low-fibre periods ($p \leq 0,05$)
≠ indicates significant weight change from low-fibre to high-fibre periods ($p \leq 0,05$).

It can be seen that 4 patients showed a small but significant reduction in weight from the uncontrolled to the low-fibre period, while only one lost weight from the low- to the high-fibre period.

The mean plasma glucose values are shown in Table 4.6 overleaf. There was no significant overall improvement in mean plasma glucose, despite the fact that patient 7 showed a significant reduction from the uncontrolled to the low-fibre diet period, and patient 18 showed a continual fall in mean plasma glucose to a very significant level during the high-fibre diet period. Patients 7 and 8, who showed the two lowest daily intakes of dietary fibre in the high-fibre period, also showed an increase in their mean plasma glucose levels during the high-fibre period.

Patient	Uncontrolled diet	Low-fibre diet	High-fibre diet
3	13,7 ± 5,8	16,7 ± 3,4	15,1 ± 3,6
4	10,8 ± 2,1	9,7 ± 2,2	8,4 ± 3,1
5	9,8 ± 1,4	11,3 ± 1,9	9,6 ± 1,7
7	16,8 ± 2,8	12,8 ± 2,3*	15,6 ± 1,3‡
8	17,3 ± 8,2	17,6 ± 2,5	22,1 ± 3,0‡
11	14,7 ± 3,1	17,0 ± 3,4	17,4 ± 3,2
13	12,5 ± 6,0	15,7 ± 6,9	15,5 ± 3,1
14	10,9 ± 4,6	14,6 ± 4,3	16,9 ± 7,7
17	21,6 ± 5,0	19,3 ± 2,9	20,8 ± 4,5
18	11,5 ± 2,8	8,4 ± 3,3	5,1 ± 2,0†

Significantly lower than uncontrolled diet: * $P < 0.025$; † $P < 0.01$.
Significantly higher than low-fibre diet: ‡ $P < 0.025$.

TABLE 4.6 Plasma Glucose (mmol/l) (Mean ± SD)

There was no significant improvement in mean HbA_1, levels throughout the study, and the total and HDL cholesterol values also showed no significant trend.

The mean serum triglyceride values are given in Table 4.7

Patient	Uncontrolled diet	Low-fibre diet	High-fibre diet
3	1,60 ± 0,36	2,02 ± 0,49	2,00 ± 0,08
4	1,59 ± 0,50	1,75 ± 0,39	2,41 ± 0,56
5	1,06 ± 0,26	1,00 ± 0,28	0,82 ± 0,26
7	1,24 ± 0,30	0,71 ± 0,17*	0,95 ± 0,06†
8	3,33 ± 1,06	2,78 ± 0,79	2,71 ± 0,17
11	1,62 ± 0,53	1,68 ± 0,31	2,02 ± 0,45
13	0,81 ± 0,22	0,84 ± 0,21	0,71 ± 0,10
14	0,98 ± 0,36	0,94 ± 0,09	0,77 ± 0,23
17	—	1,27 ± 0,30	1,08 ± 0,34
18	3,02 ± 1,06	3,93 ± 0,20	1,68 ± 0,41‡

Significantly different from uncontrolled diet: * $P < 0.025$.
Significantly different from low-fibre diet: † $P < 0.05$; ‡ $P < 0.001$.

TABLE 4.7 Serum Triglyceride (mmol/l) (Mean ± SD)

Despite the small individual changes which can be seen in the above table, in which patients 5,8 and 14 showed a trend to a decrease in their mean serum triglyceride levels throughout the study, only patient 18 showed a statistically significant decrease when the high-fibre period was compared to the low-fibre diet period.

When the low-fibre and high-fibre periods were compared, a significant negative correlation was found between the mean plasma glucose change and the dietary fibre increment, ($r = -0.704$, $p < 0.05$) as shown in figure 4.1.

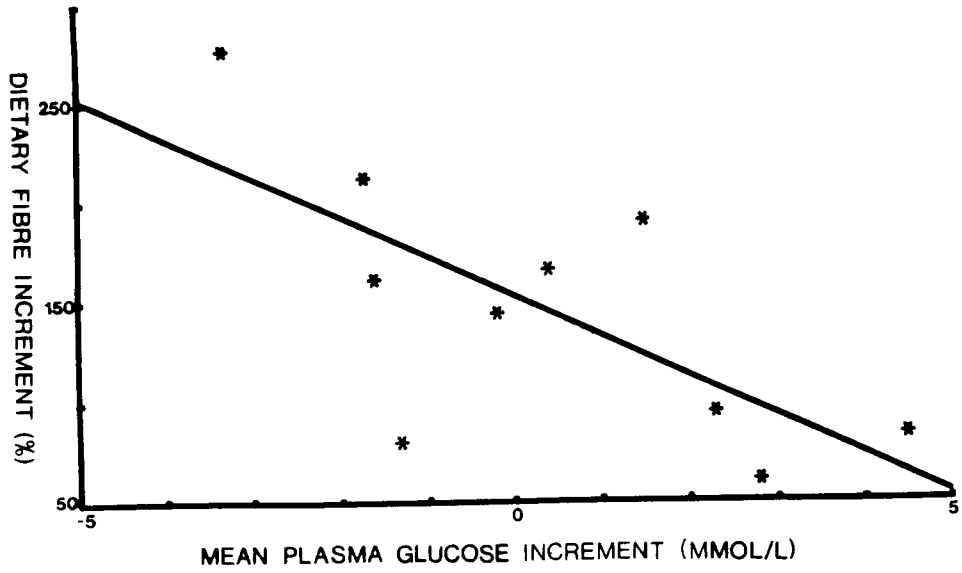

FIG. 4.1 Correlation between dietary fibre increment and mean plasma glucose levels.

Similarly, there was a significant negative correlation between the mean serum triglyceride change and the dietary fibre increment between the low-fibre and high-fibre periods ($r = -0.741$, $p < 0.05$).

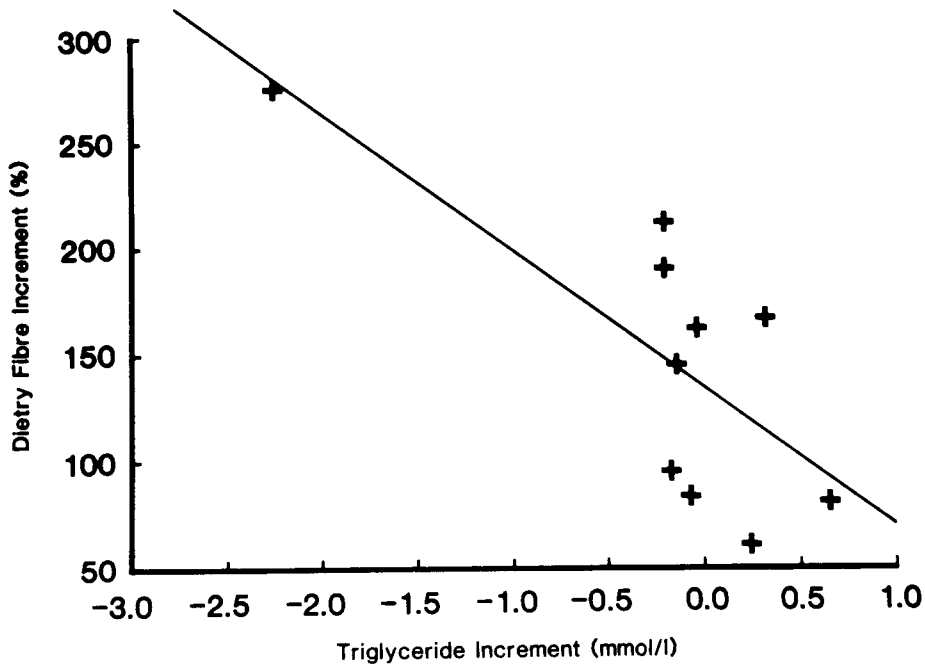

FIG. 4.2 Correlation between dietary fibre increment and triglyceride increment.

However, no correlation could be demonstrated between the increment in dietary fibre intake and the change in either HbA_1, serum cholesterol or HDL cholesterol. There was also no correlation between the difference in energy intake and the mean body weight change between the low- and high-fibre periods.

DISCUSSION

The results indicate that those patients who increased their fibre intake most, showed the greatest improvement in plasma glucose and triglyceride levels. This study thus confirms the findings published by other groups overseas, that increased dietary fibre intakes, against a background of relatively high carbohydrate intakes, lead to improved plasma glucose and serum triglyceride levels. Our findings also imply that the mean plasma glucose and triglyceride levels might have changed significantly in all patients, had they increased their dietary fibre intakes to the recommended levels. Unfortunately the majority of our patients did not attain the desired level of fibre intake, despite very close surveillance and the free supply of the high-fibre foodstuffs.

Compliance to a drastic change in basic eating habits thus apppears to be difficult to achieve. In this study we made no attempt to educate our patients as to the purpose of the trial or as to the purported effects of dietary fibre. Thus the motivation of this group of patients was not altered, other than any effect that the close supervision during the study might have had. We specifically did not wish to introduce other variables into the study by education or motivation-reward techniques, but attempted to evaluate the effect of the high-fibre foods in the situation as it existed, and their suitability for our patients, according to their life-styles.

It would therefore appear that, without adequate education and motivation to achieve an understanding of the rationale for the required change in eating habits, most of our diabetic out-patients would not be prepared to alter their diets. Consequently high-fibre foods would have little effect on improvement of diabetic control.

CONCLUSION

This study has been found to support the current concept that an increase

in dietary fibre intake, in combination with a suitable carbohydrate intake, is of benefit in improving the glycaemic control of diabetic patients. Locally-available foods have been shown to be effective in this respect, and the improvement in mean plasma glucose is proportional to the intake of dietary fibre. Furthermore, we can conclude that our ordinary run-of-the-mill patients will not adhere to a drastic change in their eating habits unless they are suitably educated and motivated to do so. Thus the practical large-scale implementation of suitable high-fibre diets will probably be difficult in our local situation. Should further studies of this nature be attempted, or should one try to introduce high-fibre diets into the routine management of diabetic patients, a comprehensive education programme will probably need to be instituted before one could hope to achieve any measure of success.

SUGGESTIONS FOR FUTURE RESEARCH

In order to test this hypothesis of the necessity for suitable education and motivation as a prerequisite in the successful use of high-fibre diets for diabetic patients, the following procedure could be followed:

Two similar groups of diabetic out-patients could be chosen, one receiving education prior to the initiation of a long-term high-fibre diet period, and the other, the control group, receiving no education prior to an identical high-fibre dietary intervention. One could then investigate the effect of education on the motivation and compliance to the high-fibre diets, by analysing blood samples and dietary records in a similar manner to that described in the above study.

CHAPTER 5

THE USE OF A GUAR SUPPLEMENT IN THE DIABETIC DIET

INTRODUCTION

Once the suggestion had been made by Trowell (1973 and 1975) that dietary fibre deficiency was a possible aetiological factor for diabetes mellitus, research began to focus on a fibre-supplemented diet as a suitable adjunct to diabetes therapy. In assessing the efficacy of a number of food sources of dietary fibre, Jenkins and his co-workers (1978a) reported that guar gum, with its viscous nature, was the fibre most effective in reducing the post-prandial response to an oral glucose tolerance test (GTT). His pioneering work, which has been described in the literature review, in incorporating guar gum into a crispbread form, obviated much of its unpalatability when it was mixed into foods as a supplement. However, side-effects were still noted with the large doses used daily (14-26g) in his studies. We therefore promoted the development of a locally-produced guar-containing biscuit which we hoped would be more acceptable in the long term as a food supplement, because of the much smaller amount of guar which it contained. Our aim was to assess firstly its efficacy in improving diabetic control, and secondly the feasibility of including this as a food supplement in the diabetic diet.

METHODS

(a) *The biscuit*

With the help of a local biscuit manufacturer, a digestive-type biscuit was produced, the composition of which is given in Table 5.1 below.

Carbohydrate (%)	**61,0**
Protein (%)	**7,0**
Fat (%)	**18,0**
Guar gum (g/100g)	**8,7**
Energy content (kJ/100g)	**1822,0**

TABLE 5.1 Composition of the guar biscuit

This biscuit was then tested against a local crispbread biscuit, "Provita", which is generally recommended for use by diabetic subjects. The composition of the Provita biscuit is given in Table 5.2 below.

Carbohydrate (%)	**75,0**
Protein (%)	**11,4**
Fat (%)	**9,3**
Bran (g/100g)	**1,8**
Energy content (kJ/100g)	**1814,5**

TABLE 5.2 Composition of Provita biscuits

(b) *The study protocols*
These were divided into 3 parts:-

Study 1
This study aimed to test the glycaemic effect of the guar biscuits against an equivalent amount of carbohydrate and energy provided by Provita biscuits and an oral GTT, as a reference standard. Nine diabetic patients, 3 IDDM and 6 NIDDM, ingested on three separate occasions, after an overnight fast and having taken no insulin or oral agent on the morning of the study, 200 ml of water with either 50g glucose, 15 provita biscuits (56,3g CHO) or 10 guar biscuits (58,0g CHO, 8,2g guar gum). 5g margarine was added to the Provita test meal in order to equalise the fat content with that of the guar biscuit meal.

Blood samples were taken from an indwelling catheter placed in the antecubital vein, before the ingestion of the test meal, and at regular intervals up to 120 minutes after ingestion. The blood was allowed to clot and the serum was assayed for glucose on an auto-analyser by means of the ferricyanide method.

Study 2
This study tested the effect of the biscuits when incorporated into the meal plan in a short-term outpatient situation, and also assessed whether any residual effect of ingesting the biscuits would occur.

Eight of the patients who had taken part in the initial study (2 IDDM and 6 NIDDM) were placed on a standard low-fibre diabetic diet for one week, consuming 5 slices of bread a day for this control period. After this, 10 guar biscuits were substituted for the bread each day for a further week

(the test period), the rest of the diet remaining unchanged. Patients kept written food records for the control period, in order to adhere to the same diet during the test period, simply exchanging the guar biscuits for bread. At the end of each week a fasting blood sample was taken and the serum glucose response to a standard test meal, containing no guar, was measured by the same methods as in Study 1.

Table 5.3 gives the composition of the test meal.

	Mass (g)	Carbo-hydrate (g)	Protein (g)	Fat (g)	Energy (kJ)
Boiled eggs (2)	**108**	**1,0**	**14,0**	**12,4**	**697,2**
Milk (250 ml)	**250**	**12,0**	**8,5**	**9,0**	**693**
Skim-milk powder (30 g)	**30**	**15,5**	**10,7**	**0,1**	**451,5**
Bread (2 slices)	**60**	**28,2**	**4,9**	**2,1**	**574,6**
Jam (2 × 20 g)	**40**	**28,0**	**0,2**	**—**	**436,8**
Butter (1 × 10 g)	**10**	**—**	**—**	**8,2**	**310,8**
Total	**598**	**84,7**	**38,3**	**31,8**	**3 163,9**
Energy provided (kJ)		**1 333,9**	**643,4**	**1 202,0**	
% energy		**42,1**	**20,2**	**37,7**	

TABLE 5.3 The Standard Test Meal

Study 3.

This study was designed to test the longer-term effects of ingesting the guar biscuits, as part of the diabetic meal plan, on the glycaemic control of diabetic outpatients. Ten patients were used, 5 on insulin therapy and 5 on oral anti-diabetic agents. Each patient was given a meal plan suited to his individual requirements and was kept on this diet for a control period of 14 days. A written food record was kept for this period. During the next 14 days the patients were given 15 guar biscuits providing 9,2g guar per day, distributed among the meals and snacks in exchange for other carbohydrates of their meal plans. The biscuits for this study were produced on a larger scale in the biscuit factory and were cut by machine to a smaller size than those used in Study 1 and 2. The rest of their diet during this period remained identical to that recorded during the control period. The patients also kept a thrice-daily record of their urinary glucose excretion, using Tes-tape (Eli Lilly) throughout the 28-day trial period, and collected a 24-hour urine on days 6, 13, 20 and 27. This was brought in

on day 7, 14, 21 and 28, and assayed for glucose, using the test for reducing sugars.

Fasting blood glucose was assayed on days 0, 7, 14, 21 and 28. Glycosylated haemoglobin (HbA_1) values were measured, by the same method as before, on days 0, 14 and 28. The patients were weighed at the beginning and end of each 14-day period.

(c) *Statistical methods*

The results were analysed using Students t-test and the Wilcoxon signed rank test where relevant. Significance was accepted at the 5% level.

(d) *Results*

Study 1.

The serum glucose response after the meal of guar biscuits, Provita biscuits and the oral glucose load, is shown in figure 5.1 below.

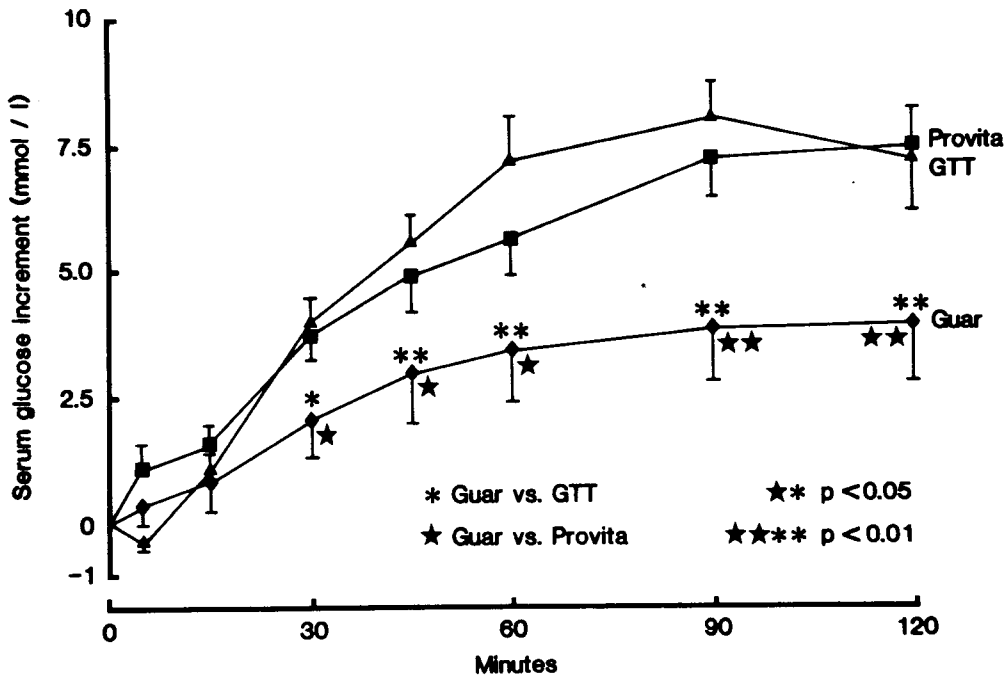

FIG. 5.1 Serum glucose responses in 9 patients (Mean ± SEM)

It will be seen that the serum glucose response after the meal of guar biscuits was significantly lower than that after the glucose load and after the Provita meal. There was no significant difference between the responses to the glucose and the Provita meal.

Study 2.

The fasting serum glucose values before and after the week on the diet containing guar biscuits, are shown in figure 5.2 below.

From figure 5.2 it will be seen that the mean serum glucose value after a week on the guar-supplemented diet was significantly lower than that after the control period ($p<0,05$). The only patient whose fasting serum glucose level was higher after the week on guar biscuits, had had to reduce his daily insulin dosage because of frequent hypoglycaemic episodes.

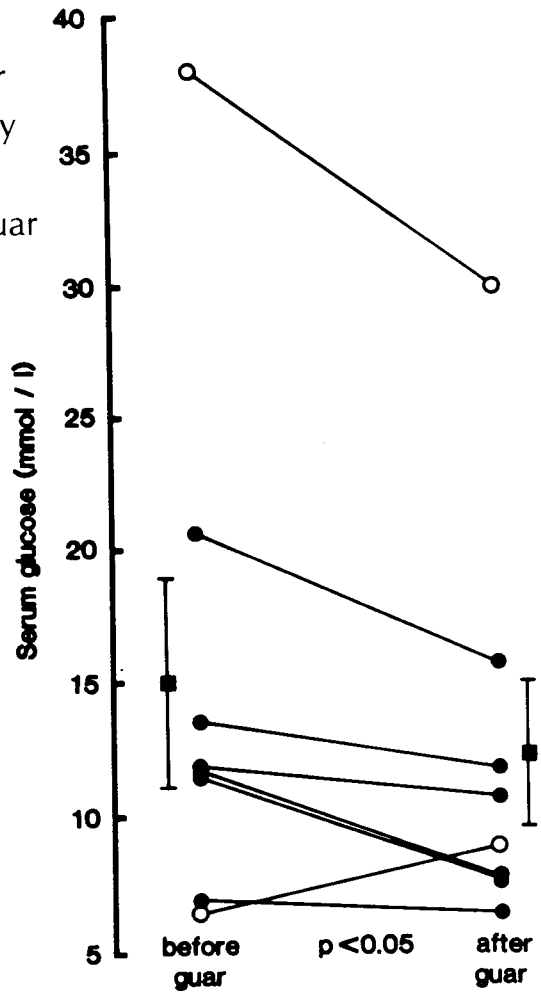

FIG. 5.2 Individual and mean (± SEM) fasting serum glucose levels in 8 patients before and after a week on a diet containing guar biscuits.
(○——○ = insulin-dependent patients)
(•——• = non-insulin-dependent patients)

Figure 5.3 shows the mean serum glucose responses to the standard test meal, given after a control week of low-fibre diabetic meal plans, and after a week on guar-supplemented meal plans.

It will be seen that the post-prandial glucose response to the standard test meal was similar on each occasion and that no significant differences were found in the serum glucose levels, besides the mean fasting level, which is the same as that shown in figure 5.2

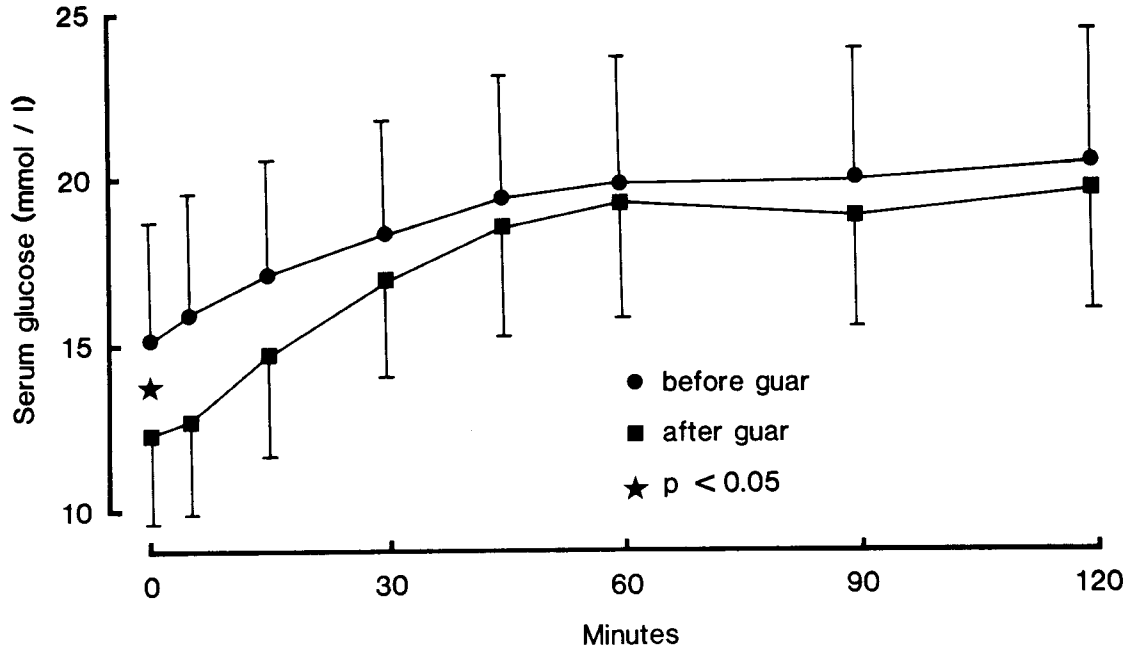

FIG. 5.3 Serum glucose responses to a mixed meal (mean ± SEM) in 8 patients before and after a week on diets containing guar biscuits.

Study 3

Results of the procedures were divided into those for IDDM and NIDDM patients. Table 5.4 gives the results for the insulin-dependent subjects.

	Body mass (kg)	Urinary glucose (mmol/24h)	HbA_1 (%)	Fasting serum glucose level (mmol/l)
Pre-study	71.8 ± 7.0	—	14.6 ± 1.1	18.4 ± 2.8
Control	72.2 ± 6.8	203.0 ± 64.5	12.6 ± 1.4‡	12.7 ± 3.5
Guar	71.0 ± 7.0*	40.1 ± 30.1*	11.2 ± 1.4‡	11.7 ± 2.9

* compared with control: $p < 0.01$
‡ compared with pre-study: $p < 0.01$

TABLE 5.4 Insulin-dependent subjects (mean ± SEM)

It will be seen that there was a highly significant reduction in 24-hour urinary glucose excretion during the period on guar gum ($p<0,01$). The mean body weight of these patients decreased significantly during the period on guar, but this difference was only slight in clinical terms. However the HbA_1 and fasting serum glucose values did not change significantly between the control and guar periods. All these patients reported an increased frequency of hypoglycaemic attacks, and two were forced to reduce their insulin doses.

The results in the non-insulin-dependent subjects are shown in Table 5.5.

	Body mass (kg)	Urinary glucose (mmol/24h)	HbA_1 (%)	Fasting serum glucose level (mmol/l)
Pre-study	76.4 ± 6.1	–	12.4 ± 1.0	9.7 ± 1.3
Control	75.3 ± 5.8⁻	14.3 ± 6.2	10.2 ± 0.75	8.5 ± 0.8
Guar	74.9 ± 6.1	5.4 ± 1.0‡	9.9 ± 0.73*	7.0 ± 1.0‡

⁻ compared with pre-study: $p < 0.01$
* compared with control: $p < 0.05$
‡ compared with control: $p < 0.01$

TABLE 5.5 Non-insulin dependent subjects (mean ± SEM)

In these patients there were highly significant reductions in 24-hour urinary glucose excretion ($p<0,01$) and mean fasting serum glucose values ($p<0,01$). The mean HbA_1 values also showed a significant fall after the period on guar biscuits. Although there was a small but significant reduction in mean body mass during the control period, no further significant reduction took place after the period on guar. Urine test records kept by both groups of patients showed a significant reduction in the number of "pluses" during the period on guar, in all patients except the two who had to reduce their insulin dose. Two specimens of such patient records are enclosed (Enclosure V).

Some patients reported mild stomach cramps or diarrhoea at the beginning of the period on the guar biscuits, but these symptoms disappeared after 3-4 days. All patients, however, were keen to continue taking the biscuits, should this be made possible.

DISCUSSION

These results in general demonstrate the potential effectiveness of guar gum, as incorporated into the biscuits, in improving diabetic control in both IDDM and NIDDM patients.

The results of Study 1 show that guar gum, when incorporated into the test biscuit, has a significant effect in blunting the post-prandial serum glucose response, and that the gum is clearly more effective than the bran fibre of Provita biscuits. This confirmsJenkins' reports on the efficacy of

guar gum when compared to other fibre sources.

It appears from Study 2, firstly, that it is possible to use the guar-containing biscuit as an exchange for other carbohydrate foods such as bread in the diabetic meal plan, and secondly that guar gum should be taken regularly as part of a meal in order to maintain its effect. Thus the gum when taken in the doses used for a week, does not seem to have any residual blood-glucose-lowering effect on subsequent non-guar-containing test meals. It did, however, improve the metabolic profile of the diabetic subjects tested, in that it lowered the mean fasting serum glucose after one week of regular use of the guar supplement. This could not have been due to any change in carbohydrate content of the test as opposed to the control period, since the biscuits were used as a substitute for other carbohydrate (bread) and not as an additional source of carbohydrate, while the rest of the meal plan remained unaltered. Thus the effect of the guar was probably mediated by its action as "lente" carbohydrate, and this slower absorption of carbohydrate into the portal blood stream might improve the diabetic control and consequently the fasting serum glucose of the patient.

This delayed absorption of carbohydrate was probably responsible for the increased frequency of hypoglycaemia reported by IDDM subjects in both Study 2 and Study 3, as well as for the significant reductions in urinary glucose excretion observed in both IDDM and NIDDM in Study 3. Although the statistical evidence of improved diabetic control seems less clear in the patients with insulin-dependent diabetes, there seemed to be a trend to improvement in fasting serum glucose and glycosylated haemoglobin. The lack of statistical proof may be at least partly due to the small number of subjects tested, and partly due to the fact that two patients in Study 3 had to reduce their insulin dose because of hypoglycaemic episodes. Hypoglycaemia is known to cause a rebound hyperglycaemia (the Somogyi effect) and this might adversely affect the levels of glycosylated haemoglobin. One would also require a trial period of longer than 4 weeks to reflect any improvement in diabetic control to a more significant degree. In the situation as tested over this two-week period on guar biscuits, we were nevertheless able to demonstrate a highly significant reduction in urinary glucose excretion in both IDDM and NIDDM subjects, and a significant reduction in all parameters of diabetic

control in NIDDM subjects after the two-week period on guar biscuits.

The biscuits were pleasant-tasting and were accepted readily by the test subjects. The initial symptoms of flatulence and diarrhoea were only transient, and could probably be avoided by gradually introducing the biscuits into the diet in increasing quantities over a period of a few days. Their acceptablity was probably due to the fact that such a small quantity of guar was incorporated into each biscuit (0,62g guar gum in the final smaller machine-produced biscuit). A dose of 3 biscuits was given with each meal in Study 3, thus providing 1,86g aliquots of guar per meal and snack. This is in great contrast to the quantities of guar used by Jenkins. Guar gum probably acts according to an all-or-none law. This in fact is in accordance with unpublished data from our group where we have found that a minimum quantity of guar gum is sufficient to lower the blood glucose response to a test meal, and that further increases in the quantity of guar given, produce no further lowering of blood glucose.

These biscuits thus appear to be a suitable vehicle for the incorporation of guar gum into a meal plan for diabetics, as an exchange for other carbohydrate foods in the diet, and regular use as such may prove to be a valuable adjunct to diabetic therapy. A much smaller dose of guar gum than has been previously reported, has been shown to be effective in lowering the post-prandial glucose reponse. This may ultimately improve all parameters of diabetic control, by delaying the absorption of nutrients, in particular carbohydrate, from the gastro-intestinal tract. This improved diabetic control may be easier to achieve in NIDDM patients, but it may also be possible to effect an improvement in IDDM patients, with careful monitoring of their insulin regimes, and perhaps with anticipatory reduction in insulin dosage as the guar supplement is introduced.

Use of this guar supplement may thus mean that the reduction of the dose of other anti-diabetic agents becomes possible, which would probably have a beneficial effect on the mental attitude of the patient. As his diabetic control would appear to be improving, he might feel more confident in his ability to treat his disease and this in turn could improve his compliance generally to all therapeutic recommendations.

CHAPTER 6

CONCLUSIONS

The achievement of good glycaemic control of diabetes mellitus is the essential goal of current therapy for this disease. This can only occur provided that four factors act synergistically:

suitable medication to control the metabolic abnormalities;
education of the patient in self-care techniques;
the devising of an individualised "diet" or meal plan to normalise glycaemic excursions;
compliance of the patient to all therapeutic recommendations.

The purpose of this thesis was to assess various methods, either educational or dietary, for their efficacy in improving the glycaemic control of diabetic out-patients, and the role of compliance in achieving this control.

From the educational project (Chapter 3) one can conclude that it is extremely difficult to improve diabetic control by means of a programme which increases the patients' general knowledge of diabetes. Although it was possible to increase, to a highly significant degree, patients' knowledge of the disease and the rules of the diabetic diet, compliance to dietary or medication procedures did not change, nor did the standard of diabetic control. Lack of compliance to the "diabetic diet", despite increased dietary knowledge, may have been one of the factors which prevented an improvement in glycaemic control. In a mass-education programme it is difficult to reach the individual patient, and motivate him to change his eating habits, or to assess reasons for non-adherence to the diet. A one-to-one method of dietary instruction may prove more effective here. Other factors which may have been relevant in non-compliance could be the personality of the patient, communication with the doctor, patient satisfaction and the patients' concept of the severity of his disease.

Nevertheless, statistical analysis of our data supports the published reports that improved knowledge of diabetes is not necessarily associated with improved control. Furthermore, we have shown that the standard of

control of diabetes was not associated either with age of the patient, or with knowledge scores, but that in fact patient knowledge deteriorated with age.

The long-term high-fibre dietary study (Chapter 4) also shows that compliance to a required alteration in eating habits is very poor. Here, although improvement of diabetic control was associated with increased dietary fibre intakes, very few patients approached the recommended level of fibre intake, and none achieved it. One can thus conclude that locally-available high-fibre foodstuffs would be effective in improving diabetic control, provided that patients were prepared to change their eating habits drastically, in order to ingest sufficient quantities of fibre. Long-term compliance (i.e. for the life-long duration of the disease) would be even more difficult to achieve. Possibly only extremely well-motivated, educated patients would be prepared to do this.

The use of supplements of viscous fibre in the diabetic diet (Chapter 5) appears by comparison with the other methods tested in this thesis to be the most effective and practicable means of achieving an improvement in diabetic control. No special education was required, nor was any drastic change in eating habits necessary, and compliance to this regimen presented no problem in the patients tested. More large-scale and longer-term studies should of course be undertaken, but the guar-containing biscuit has been shown to be effective in reducing post-prandial glycaemic excursions, and appears to be a palatable means of incorporating guar gum into the diabetic diet. It is thus a practical application of current principles of dietary therapy for diabetes, by providing a high-fibre, slowly absorbed form of carbohydrate.

I therefore suggest that, although education of the diabetic patient is an essential part of his therapy, it would be easier to achieve good glycaemic control by means of a palatable guar supplement than by a drastic change in eating habits in order to include the large amounts of dietary fibre necessary to benefit blood glucose levels.

APPENDIX

STATISTICAL METHODS FOR STUDY 1.

Analysis of the data was carried out by means of a set of programmes entitled "The Biomedical Computer Programme". This provided various correlations such as the Chi-squared test, the contingency coefficient and the Kendall's Tau-B correlation coefficient for testing dependence between the variables. The Mann-Whitney U-test was used for comparison of the two independent samples. Significance was accepted at the 5% ($p \leq 0,05$) level. Histogram plots and univariate summaries were also run for each variable.

For the Chi-squared test, a large value indicates significant association, together with a p value of $\leq 0,05$.

For the contingency coefficient and the Kendall's Tau-B statistic, a value close to 0 indicates no association, while positive or negative values from 0,2 and approaching $\pm$ 1 indicate increasingly significant association.

The measures of correlation by means of the contingency coefficient and the Kendall's Tau-B correlation coefficient, have been found by Hastie (1978) to be the best correlation tests for the situation as in Study 1, where two independent samples are compared.

BIBLIOGRAPHY

AMERICAN DIABETES ASSOCIATION (1979): Principles of nutrition and dietary recommendations for individuals with diabetes mellitus. Diabetes 28: 1027.

ANDERSON, J.W. (1977): Effect of carbohydrate restriction and high-carbohydrate diets on men with chemical diabetes. Am. J. Clin. Nutr. 30: 402.

ANDERSON, J.W. (1980): The role of dietary carbohydrate and fibre in the control of diabetes. Adv. Int. Med. 26: 67.

ANDERSON, J.W. & WARD, K. (1978): Long-term effects of high-carbohydrate, high-fibre diets on glucose and lipid metabolism. Diab. Care 1 (2): 77.

ANDERSON, J.W. & WARD, K. (1979): High-carbohydrate, high-fibre diets for insulin-treated men with diabetes mellitus. Am. J. Clin. Nutr. 32: 2312.

ANDERSON, J.W. & CHEN, W.L. (1979): Plant fibre. Carbohydrate and lipid metabolism. Am. J. Clin. Nutr. 32: 346.

ANDERSON, J.W., MIDGLEY, W. & WEDMAN, B. (1979): Fibre and Diabetes. Diab. Care 2: 369.

ANDERSON, J.W., FERGUSON, S., KAROUNOS, D., et al. (1980): Mineral and vitamin status on high-fibre diets. Diab. Care 3(1): 38.

ANTONIS, A. & BERSOHN, I. (1961): The influence of diet on serum triglycerides. Lancet 280(1): 3.

APLING, E.C., LEEDS, A., WOLEVER, T., et al. (1977): How to make guar bread. Lancet 2: 975.

ARKY, R., WYLIE-ROSETT, J. & EL-BEHERI, B. (1982): Examination of current dietary recommendations for individuals with diabetes mellitus. Diab. Care 5: 59.

ARO, A., UUSITUPA, M., VOUTILAINEN, E., et al (1981): Improved control and hypocholesterolaemic effect induced by long-term dietary supplementation with guar gum in Type 2 (insulin-independent) diabetes. Diabetologia 21: 29.

BANTLE, J.P., LAINE, D., CASTLE, G., et al (1983): Postprandial glucose

and insulin responses to meals containing different carbohydrates in normal and diabetic subjects. N. Eng. J. Med. 309: 7.
BEASER, S.B. (1956): Teaching the diabetic patient. Diabetes 5: 146.
BEGGAN, M.P., CREGAN, D. & DRURY, M. (1982): Assessment of the outcome of an education programme of diabetes self-care. Diabetologia 23: 246.
BOSELLO, O., OSTUZZI, R., ARMELLINI, F., et al (1980): Glucose tolerance and blood lipids in bran-fed patients with impaired glucose tolerance. Diab. Care 3: 46.
BREIDAHL, H.D. (1980): What patients with diabetes want to know about their therapy. Drugs 19: 135.
BRODY, D.S. (1980): An analysis of patient recall of their therapeutic regimens. J. Chron. Dis. 33: 57.
BRUNZELL, J.D., LERNER, R.L., HAZARD, W., et al. (1971): Improved glucose tolerance with high carbohydrate feeding in mild diabetes. N. Eng. J. Med. 284: 521.
BRUNZELL, J.D., LERNER, R.L., PORTE, D., et al. (1974): Effect of a fat free, high carbohydrate diet on diabetic subjects with fasting hyperglycaemia. Diabetes 23: 138.
BUCHANAN, N., SHUENYANE, E., MASHIGO, S., et al. (1979): Factors influencing drug compliance in ambulatory black urban patients. S. Afr. Med. J. 55(10): 368.
CAHILL, J.F. (1976): "Control" and diabetes. N.Eng. J. Med. 294: 1004.
CARLSON, L.A. & BÖTTIGER, L.E., (1972): Ischaemic heart disease in relation to fasting values of plasma triglycerides and cholesterol. Lancet 1: 865.
CERKONEY, K.A.B. & HART, L.K. (1980): The relationship between the health belief model and compliance of persons with diabetes mellitus. Diab. Care 3: 594.
CHANDALIA, H.B. & BAGRODIA, J. (1979): Effect of nutritional counselling on the blood glucose and nutritional knowledge of diabetic subjects. Diab Care 2: 353.
CHRISTIANSEN, J.S., BONNEVIE-NIELSEN, V., SVENDSEN, P., et al (1980): Effect of guar gum on 24-hour insulin requirements of insulin-dependent diabetic subjects as assessed by an artificial pancreas. Diab.

Care 3: 659.
COHEN, M., LEONG, V., SALMON, E., et al (1980): Role of guar and dietary fibre in the management of diabetes mellitus. Med. J. Austr. 1: 59.
COLLINGS, P., WILLIAMS, C. & MacDONALD, I. (1981): Effects of cooking on serum glucose and insulin responses to starch. Br. Med. J. 282: 1032.
COULSTON, A., GREENFIELD, M. KRAEMER, F., et al. (1980): Effect of source of dietary carbohydrate on plasma glucose and insulin responses to test meals in normal subjects. Am. J. Clin. Nutr. 33: 1279.
CRAPO, P.A., REAVEN, G., OLEFSKY, J. (1976): Plasma glucose and insulin responses to orally administered simple and complex carbohydrates. Diabetes 25: 741.
CRAPO, P.A., REAVEN, G. & OLEFSKY, J. (1977): Post-prandial plasma-glucose and -insulin responses to different complex carbohydrates. Diabetes 26: 1178.
CRAPO, P.A., KOLTERMAN, O., WALDECK, N., et al (1980): Postprandial hormonal responses to different types of complex carbohydrate in individuals with impaired glucose tolerance. Am. J. Clin. Nutr. 33: 1723.
CRAPO, P.A. & OLEFSKY, J.M. (1983): Food fallacies and blood sugar. N. Eng. J. Med. 309: 44.
CROFFORD, O.B. (1976): Report of the National Commission on Diabetes, Vol. 1. U.S.D.H.E.W. Publication, Washington, D.C.
DAHLBERG, G., JORPES, E., KALLNER, S., et al. (1947): Diabetes mellitus in Sweden. Acta Med. Scandin. Suppl. 188.
DE BOUT, A.J., BAKER, I.A., ST. LEGER, A.S., et al (1981): A randomised controlled trial of the effect of low fat diet advice on dietary response in insulin independent diabetic women. Diabetologia 21: 529.
EASTWOOD, M.A. & KAY, R.M. (1979): An hypothesis for the action of dietary fibre along the gastro-intestinal tract. Am. J. Clin. Nutr. 32: 364.
ESTRICH, D., RAVNICK, A., SCHLIERF, G., et al (1967): Effects of co-ingestion of fat and protein upon carbohydrate-induced hyperglycaemia. Diabetes 16: 232.
ETZWILER, D.D. (1972): The patient is a member of the medical team.

J. Am. Diet. Assoc. 61: 421.
ETZWILER, D.D. (1978): Education of the patient with diabetes. Med. Clin. N. Am. 62: 857.
ETZWILER, D.D. & MAIMAN, L.A. (1982), *in* Diabetes Mellitus and Obesity; ed. Brodoff, B.N. and Bleicher, S.J., Williams and Wilkins, Baltimore.
FARQUHAR, J.W. FRANK, A., GROSS, R., et al (1966): Glucose, insulin and triglyceride response to high and low carbohydrate diets in man. J. Clin. Invest. 45: 1648.
GIBSON, R.B. (1929): Latent tolerance in diabetes mellitus. J. Lab. Clin. Med. 14: 597.
GREY, N. & KIPNIS D.M. (1971): Effect of diet composition on the hyperinsulinaemia of obesity. N. Eng. J. Med. 285: 827.
HABER, G.B., HEATON, K., MURPHY, D., et al (1977): Depletion and disruption of dietary fibre:- effects on satiety, plasma-glucose and serum-insulin. Lancet 2: 679.
HADDEN, D.R., MONTGOMERY, D.A., SKELLY, R.J., et al. (1975): Maturity onset diabetes mellitus: response to intensive dietary management. Br. Med. J. 3: 276.
HALL, S.E., BOLTON, T. & HETENYI, G. (1980): The effect of bran on glucose kinetics and plasma insulin in non-insulin-dependent diabetes mellitus. Diab. Care 3: 520.
HASSELL, J. & MEDVED, E. (1975): Group/audiovisual instruction for patients with diabetes. J. Am. Diet. Assoc. 66: 465.
HASTIE, T.J. (1978): Thesis submitted in fulfilment of the requirements for the degree of M.Sc. (Maths. Stats., U.C.T.)
HAYNES, R.B., TAYLOR, D. & SACKETT, D. (1979) eds: Compliance in Health Care. Johns Hopkins Univ. Press, Baltimore.
HIMSWORTH, H.P. (1933): The physiological activation of insulin. Clin. Sci. 1: 1.
HIMSWORTH, H.P. (1935): The dietetic factor determining the glucose tolerance and sensitivity to insulin of healthy men. Clin. Sci. 2: 67.
HIMSWORTH, H.P. (1949): The syndrome of diabetes mellitus and its causes. Lancet 1: 465.
HIMSWORTH, H.P. & KERR, R.B. (1939): Insulin-sensitive and insulin-

insensitive types of diabetes mellitus. Clin. Sci. 4: 119.
HOCKADAY, T.D.R., HOCKADAY, J., MANN, J., et al. (1978): Prospective comparison of modified-fat high-carbohydrate with standard low-carbohydrate dietary advice in the treatment of diabetes. Br. J. Nutr. 39: 357.
HOFFMANN, C.R., FINEBERG, S., HOWEY, D., et al. (1982): Short-term effects of a high-fibre high-carbohydrate diet in very obese diabetic individuals. Diab. Care 5: 605.
HOLLAND, W.M. (1968): The diabetes supplement of the National Health Survey. J. Am. Diet. Assoc. 52: 387.
HOLT, S., HEADING, R., CARTER, D., et al (1979): Effect of gel fibre on gastric emptying and absorption of glucose and paracetamol. Lancet 1: 636.
HULKA, B.S. (1976): Communication, compliance, and concordance between physicians and patients with prescribed medications. Am. J. Pub. Health 66: 847.
IONESCU-TIRGOVISTE, C., POPA, E., SINTU, E., et al. (1983): Blood glucose and plasma insulin responses to various carbohydrates in Type 2 (non-insulin-dependent) diabetes. Diabetologia 24: 80.
JACKSON, W.P.U., KALK, J., HARDCASTLE, A., et al (1972): Glucose intolerance retested. S. Afr. Med. J. 46: 2065.
JACKSON, W.P.U. & VINIK, A.I. (1977): Diabetes Mellitus, Clinical and Metabolic. Juta & Co., Cape Town.
JENKINS, D.J.A. (1979): Dietary fibre, diabetes and hyperlipidaemia. Lancet 2: 1287.
JENKINS, D.J.A. (1981a): Lack of effect of refining on the glycaemic response to cereals. Diab. Care 4: 509.
JENKINS, D.J.A. (1981b): Glycaemic index of foods: a physiological basis for carbohydrate exchange. Am. J. Clin. Nutr. 34: 362.
JENKINS, D.J.A. (1982): Lente carbohydrate: a newer approach to the dietary management of diabetes. Diab. Care 5: 634.
JENKINS, D.J.A., GOFF, D., LEEDS, A., et al (1976): Unabsorbable carbohydrates and diabetes: decreased post-prandial hyperglycaemia. Lancet 2: 172.
JENKINS, D.J.A., LEEDS, A., GASSULL, M., et al (1977a): Decrease in

post-prandial insulin and glucose concentrations by guar and pectin. Ann. Int. Med. 86: 20.

JENKINS, D.J.A., WOLEVER, T., HOCKADAY, T., et al (1977b): Treatment of diabetes with guar gum. Lancet 2: 779.

JENKINS, D.J.A., WOLEVER, T., LEEDS, A., et al. (1978a): Dietary fibres, fibre analogues and glucose tolerance: importance of viscosity. Br. Med. J. 1: 1392.

JENKINS, D.J.A., WOLEVER, T., NINEHAM, R., et al (1978b): Guar crispbread in the diabetic diet. Br. Med. J. 2: 1744.

JENKINS, D.J.A., WOLEVER, T., NINEHAM, R., et al. (1980a): Improved glucose tolerance 4 hours after taking guar with glucose. Diabetologia 19: 21.

JENKINS, D.J.A., WOLEVER, T., TAYLOR, R., et al (1980b): Diabetic glucose control, lipids and trace elements on long-term guar. Br. Med. J. 280: 1353.

JENKINS, D.J.A., WOLEVER, T., BACON, S., et al. (1980c): Diabetic diets: high carbohydrate combined with high fibre. Am. J. Clin. Nutr. 33: 1729.

JENKINS, D.J.A., WOLEVER, T., TAYLOR, R., et al (1980d): Exceptionally low blood glucose response to dried beans: comparison with other carbohydrate foods. Br. Med. J. 281: 578.

JENKINS, D.J.A., WOLEVER, T., TAYLOR, R., et al (1980e): Rate of digestion of foods and post-prandial glycaemia in normal and diabetic subjects. Br. Med. J. 281: 14.

JENKINS, D.J.A. & WOLEVER, T.M.S. (1981): Slow release carbohydrate and the treatment of diabetes. Proc. Nutr. Soc. 40: 227.

JENKINS, D.J.A., GHAFARI, H., WOLEVER, T., et al (1982a): Relationship between rate of digestion of foods and post-prandial glycaemia. Diabetologia 22: 450.

JENKINS, D.J.A., THORNE, M.J., CAMELON, K., et al (1982b): Effect of processing on digestibility and the blood glucose response: a study of lentils. Am. J. Clin. Nutr. 36: 1093.

JENKINS, D.J.A., WOLEVER, T., TAYLOR, R., et al (1982c): Slow release dietary carbohydrate improves second meal tolerance. Am. J. Clin. Nutr. 35: 1339

JENKINS, D.J.A., WOLEVER, T., JENKINS, A., et al (1983a): Glycaemic response to wheat products; reduced response to pasta but no effect of fibre. Diab. Care 6: 155.

JENKINS, D.J.A., WOLEVER, T., JENKINS, A., et al (1983b): The glycaemic index of foods tested in diabetic patients; a new basis for carbohydrate exchange favouring the use of legumes. Diabetologia 24: 257.

KAHN, S.E. & KLAFF, L.J. (1983): The importance of metabolic control in diabetes mellitus. Cont. Med. Ed. 1: 15.

KATO, H., TILLOTSON, J., NICHAMAN, M.Z., et al (1973): Epidemiological studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii and California. Am. J. Epidemiol. 97: 372.

KAUFMAN, M. (1964): The many dimensions of diet counselling for diabetes. Am. J. Clin. Nutr. 15: 45.

KAY, R.M., GROBIN, W. & TRACK, N. (1981): Diets rich in natural fibre improve carbohydrate tolerance in maturity-onset, non-insulin dependent diabetics. Diabetologia 20: 18.

KELSAY, J.L., BEHALL, K.M. & PRATHER, E.S. (1978): Effect of fibre from fruits and vegetables on metabolic responses of human subjects. Am. J. Clin. Nutr. 31: 1149.

KIEHM, T.G., ANDERSON, J. & WARD, K. (1976): Beneficial effects of a high carbohydrate, high fibre diet on hyperglycaemic diabetic men. Am. J. Clin. Nutr. 29: 895.

KINMONTH, A.L., ANGUS, R.M., JENKINS, P.A., et al (1982): Whole foods and increased dietary fibre improve blood glucose control in diabetic children. Arch. Dis. Childh. 57: 187.

KUHL, C., MØLSTED-PEDERSEN, L., HORNNES, P.J., et al (1983): Guar gum and glycaemic control of pregnant insulin-dependent diabetic patients. Diab. Care 6: 152.

KYNOCH, P.A. & LEHMANN, H. (1977): Rapid estimation (2½ hours) of glycosylated haemoglobin for routine purposes. Lancet 2: 16.

LAWRENCE, P.A. & CHEELY, J. (1980): Deterioration of diabetic patients' knowledge and management skills as determined during outpatient visits. Diab. Care 3: 214.

LAWRENCE, R.D. & MCCANCE, R.A. (1929): New analysis of

carbohydrate foods and their application to diabetic diets. Brit. Med. J. 2: 241.

LEICHTER, S.B. & CHANDLER, C.A. (1982) *in* Diabetes Mellitus and Obesity. eds. Brodoff, B.N. and Bleicher, S.J. Williams and Wilkins, Baltimore.

LENNER, R.A. (1976): Studies of glycaemia and glycosuria in diabetics after breakfast meals of different composition. Am. J. Clin. Nutr. 29: 716.

LEVITT, N.S., VINIK, A., SIVE, A., et al (1980): The effect of dietary fibre on glucose and hormone responses to a mixed meal in normal subjects and in diabetic subjects with and without autonomic neuropathy. Diab. Care 3: 515.

LINN, M.W., LINN, B., SKYLER, J., et al (1980): The importance of self-assessed health in patients with diabetes. Diab. Care 3: 599.

LOPES-VIRELLA, M.F., WOHLTMANN, H.J., LOADHOLT, C.B., et al (1981): Plasma lipids and lipoproteins in young insulin-dependent patients; relationship with control. Diabetologia 21: 216.

LOWERY, B.J. (1976): Disease-related learning and disease control in diabetics as a function of locus of control. Nurs. Res. 25: 358.

LUDVIGSON, J., RICHT, B. & SVENSSON, P. (1980): Compliance and the health-belief model. Scand. J. Soc. Med. 18 (Suppl): 58.

MANN, J. (1980): Diet and diabetes. Diabetologia 18: 89.

MEDALIE, J. (1979): Risk factors other than hyperglycaemia in diabetic macrovascular disease. Diab. Care 2: 77.

MIETTINEN, M., TURPEINEN, O., KARVONEN., et al (1972): Effect of cholesterol-lowering diet on mortality from coronary heart disease and other causes. Lancet 2: 835.

MILLER, L.V., GOLDSTEIN, J. & NICOLAISEN, G. (1978): Evaluation of patients' knowledge of diabetes self-care. Diab. Care 1: 275.

MIRANDA, P.M. & HORWITZ, D.L. (1978): High-fibre diets in the treatment of diabetes mellitus. Ann. Int. Med. 88: 482.

MONNIER, L.H., PHAM, T., AGUIRRE, L., et al (1978): Influence of indigestible fibres on glucose tolerance. Diab. Care 1: 83.

MONNIER, L.H., BLOTMAN, M., COLETTE, C., et al (1981): Effects of dietary fibre supplementation in stable and labile insulin-dependent

diabetics. Diabetologia 20: 12.
MORGAN, L.M., GOULDER, T., TSIOLAKIS, D., et al (1979): The effect of unabsorbable carbohydrate on gut hormones. Diabetologia 17: 85.
MOYNAHAN, E.J. (1977): Nutritional hazards of a high-fibre diet. Lancet 1: 654.
MUNOZ, J.M., SANDSTEAD, H., & JACOB, R. (1979): Effects of dietary fibre on glucose tolerance of normal men. Diabetes 28: 496.
NUTTALL, F.Q. (1980): Dietary recommendations for individuals with diabetes mellitus, 1979. Am. J. Clin. Nutr. 33: 1311.
O'CONNOR, N., TREDGER, J., MORGAN, L. (1981): Viscosity differences between various guar gums. Diabetologia 20: 612.
O'DEA, K., NESTEL, P., ANTONOFF, L. (1980): Physical factors influencing post-prandial glucose and insulin responses to starch. Am. J. Clin. Nutr. 33: 760.
OLIVER, M.F. (1981): Diet and Coronary Heart Disease. Brit. Med. Bull. 37: 49.
PAGE, P., VERSTRAETE, D., ROBB, J. et al (1981): Patient recall of self-care recommendations in diabetes. Diab. Care 4: 96.
PAUL, A.A. & SOUTHGATE, D.A.T. (1978): The composition of foods. 4th ed. revised. London. H.M.S.O.
PEDERSEN, O., HJØLLUND, E., LINDSKOV, H.O., et al (1982): Increased insulin receptor binding to monocytes from insulin-dependent diabetic patients after a low-fat, high-starch, high-fibre diet. Diab. Care 5: 284.
PETERSON, C.M., FORHAM, S.E., & JONES, R.L. (1980): Self-management; an approach to patients with insulin-dependent diabetes mellitus. Diab. Care 3: 82.
RABINOWITCH, I.M. (1935): Effects of the high carbohydrate – low calorie diet upon carbohydrate tolerance in diabetes mellitus. Canad. Med. Assoc. J. 33: 136.
RABKIN, S.W., BOYKO, E., WILSON, A.A., et al (1983): A randomised clinical trial comparing behaviour modification and individual counselling in the nutritional therapy of non-insulin-dependent diabetes mellitus. Diab. Care 6: 50.
RAY, T.K., MANSELL, K.M., KNIGHT, L.C., et al (1983): Long-term effects of dietary fibre on glucose tolerance and gastric emptying in non-

insulin-dependent diabetic patients. Am. J. Clin. Nutr. 37: 376.
REAVEN, G.M. (1981): How much carbohydrate? Diabetologia 20: 508.
REISER, S., HALLFRISCH, J., MICHAELIS, O., et al (1979): Isocaloric exchange of dietary starch and sucrose in humans. Am. J. Clin. Nutr. 32: 1659.
RICHARDSON, R. (1929): High-carbohydrate diets in diabetes mellitus. Am. J. Med. Sci. 177: 426.
RIVELLESE, A., RICCARDI, G., GIACCO, A., et al (1980): Effect of dietary fibre on glucose control and serum lipoproteins in diabetic patients. Lancet 2: 447.
ROBERTS, M.C. & WURTELE, S.K. (1980): On the non-compliant research subject in a study of medical non-compliance. Soc. Sci. & Med. 14A: 171.
RUDNICK, P.A. & TAYLOR, K.W. (1965): Effect of prolonged carbohydrate restriction on serum insulin levels in mild diabetes. Brit. Med. J. 1: 1225.
SANSUM, W.D., BLATHERWICK, N.R., & BOWDEN, R. (1926): The use of high carbohydrate diets in the treatment of diabetes mellitus. J. Am. Med. Assoc. 86: 178.
SCHAUBERGER, G., BRINCK, U., GULDNER, G. et al (1977): Exchange of carbohydrates according to their effect on blood glucose. Diabetes 26: 415 (A).
SHEKELLE, R.B., SHRYOCK, A., OGLESBY, P. et al (1981): Diet, serum cholesterol and death from coronary heart disease. N. Eng. J. Med. 304: 65.
SIMPSON, R.W., MANN, J.I., HOCKADAY, T.D.R. et al (1979a): Lipid abnormalities in untreated maturity-onset diabetics and the effect of treatment. Diabetologia 16: 101.
SIMPSON, R.W., MANN, J., EATON, J., et al (1979b): Improved glucose control in maturity-onset diabetes treated with high-carbohydrate-modified fat diet. Br. Med. J. 1: 1753.
SIMPSON, R.W., MANN, J., EATON, J. et al (1979c): High-carbohydrate diets and insulin-dependent diabetics. Br. Med. J. 2: 523.
SIMPSON, H.C.R., SIMPSON, R., LOUSLEY, S. et al (1981): A high carbohydrate leguminous fibre diet improves all aspects of diabetic

control. Lancet 1: 1.
SIMPSON, H.C.R., CARTER, R., LOUSLEY, S. et al (1982): Digestible carbohydrate – an independent effect on diabetic control in Type 2 (non-insulin depedent) diabetic patients? Diabetologia 23: 235.
SINGH, I. (1955): Low-fat diet and therapeutic doses of insulin in diabetes mellitus. Lancet 268: 422.
SKYLER, J.S., ELLIS, G.J., SKYLER, D.L. et al (1979): Instructing patients in altering insulin dosage. Diab. Care 2: 39.
SOUTHGATE, D.A.T. (1973): Fibre and the other unavailable carbohydrates and their effects on the energy value of the diet. Proc. Nutr. Soc. 32: 131.
SOUTHGATE, D.A.T. (1976): A guide to calculating intakes of dietary fibre. J. Hum. Nutr. 30: 303.
SOUTHGATE, D.A.T., PAUL, A.A., DEAN, A.G. et al (1978): Free sugars in foods. J. Hum. Nutr. 32: 335.
STONE, D.B. (1961): A study of the incidence and causes of poor control in patients with diabetes mellitus. Am. J. Med. Sci. 241: 436.
STONE, D.B. & CONNOR, W.E. (1963): The prolonged effects of a low-cholesterol, high carbohydrate diet upon the serum lipids in diabetic patients. Diabetes 12: 127.
STOWERS, J.M. (1963): Nutrition in Diabetes. Nutr. Abstr. Rev. 33: 1.
SULWAY, M., TUPLING, H., WEBB, K. et al (1980): New techniques for changing compliance in diabetes. Diab. Care 3: 108.
SURWIT, R.S., SCOVERN, A.W., & FEINGLOS, H.N. (1982): The role of behaviour in diabetes care. Diab. Care 5: 337.
SWAN, D.C., DAVIDSON, P., ALBRINK, M.J. et al (1966): Effect of simple and complex carbohydrates on plasma non-esterified fatty acids, plasma-sugar and plasma-insulin during oral carbohydrate-tolerance tests. Lancet 1: 60.
SWEENEY, J.S. (1927): Dietary factors that influence the dextrose tolerance test. Arch. Int. Med. 40: 818.
TROWELL, H.C. (1973): Dietary fibre, ischaemic heart disease and diabetes mellitus. Proc. Nutr. Soc. 32: 151.
TROWELL, H.C. (1975): Dietary fibre hypothesis of the aetiology of diabetes mellitus. Diabetes 24: 762.

TROWELL, H.C., SOUTHGATE, D.A.T., WOLEVER, T.M.S., et al (1976): Dietary fibre redefined. Lancet 1: 967.
TUNBRIDGE, R.E. (1953): Socio-medical aspects of diabetes mellitus. Lancet 265: 893.
TUNBRIDGE, R.E. & WETHERILL, J.H. (1970): Reliability and cost of diabetic diets. Br. Med. J. 2: 78.
VAN SOEST, P.J. & ROBERTSON, J.B. (1977): What is fibre and fibre in food? Nutr. Rev. 35: 12.
WAHLQUIST, M.L., MORRIS, M., LITTLEJOHN, G., et al (1979): The effects of dietary fibre on glucose tolerance in healthy males. Aust. N.Z.J. Med. 9: 154.
WALKER, A.R.P. (1961): Crude fibre, bowel motility and pattern of diet. S. Afr. Med. J. 35: 114.
WALKER, A.R., WALKER, B.F., RICHARDSON, B.D., et al (1970): Glucose and fat tolerance in Bantu children. Lancet 2: 51.
WALL, J.R., PYKE, D.A. & OAKLEY, W.G. (1973): Effect of carbohydrate restriction in obese diabetics. Br. Med. J. 1: 577.
WAPNICK, S., WICKS, A.C.B., KANENGONI, E., et al (1972): Can diet be responsible for the initial lesion in diabetes? Lancet 2: 300.
WEST, K.M. (1973): Diet therapy of diabetes: an analysis of failure. Ann. Int. Med. 79: 425.
WEINSIER, R.L., SEEMAN, A., HERRERA, M., et al (1974): High- and low-carbohydrate diets in diabetes mellitus. Ann. Int. Med. 80: 332.
W.H.O. (1970): Thirteenth report of Joint FAO/WHO Expert Committee on Food Additives, Rome.
W.H.O. (1975): Nineteenth report of Joint FAO/WHO Expert Committee on Food Additives, Geneva.
WILLIAMS, D.R.R., JAMES, W. & EVANS, I. (1980): Dietary fibre supplementation of a 'normal' breakfast administered to diabetics. Diabetologia 18: 379.
WILLIAMS, T.F. (1967): The clinical picture of diabetic control studied in four settings. Am. J. Pub. Health 57: 441.
WILLIAMS, T.F., ANDERSON, E., WATKINS, J.D., et al (1967): Dietary errors made at home by patients with diabetes. J. Am. Diet. Assoc. 51: 19.

PAPERS PUBLISHED AND TALKS PRESENTED AS A RESULT OF THE PREPARATION OF THIS THESIS

1. Smith, C.J., Rosman, M.S., Levitt, N.S. et al (1982): Guar biscuits in the diabetic diet. S. Afr. Med. J. 61: 196.

2. Rosman, M.S., Smith, C.J. and Jackson W.P.U. (1983): The effect of long-term high-fibre diets in diabetic outpatients. S. Afr. Med. J. 63: 310.

3. Smith, C.J. (1983): Meal planning for the diabetic. S. Afr. J. of Contin. Med. Ed. 1: 31.

4. Smith, C.J., Rosman, M.S., Henshilwood, P.A. et al (1982): The effect of a mass-education programme on compliance and control in diabetic out-patients. Presented at the Symposium on Current Trends in Diabetes Mellitus, Cape Town.

5. Smith, C.J., Rosman, M.S., Levitt, N.S. et al (1983): The use of guar gum in the diabetic diet. Presented at the International Conference on Diet and Nutrition, Tel Aviv, Israel.

6. Smith, C.J. (1983): Education of the Diabetic Patient, and The Diabetic Diet. Both presented at the 54th Congress of MASA, Cape Town.

ENCLOSURE 1

Dear Patient,

We want to find out how much you know about your diabetes. Please answer honestly - this will help us to help you. Everything will be strictly confidential and used for research purposes only.
Thank you for your co-operation.

Mrs. C. Smith

Geagte Pasient,

Ons wil graag uitvind hoeveel u van u suikersiekte weet. Beantwoord asseblief alle vrae eerlik - dit sal ons help om u te help. Alles sal streng vertroulik gehou word, en is slegs vir navorsingsdoeleindes.
Baie dankie vir u samewerking.

Mev. C. Smith

DIABETES OUTPATIENT CLINIC - GROOTE SCHUUR HOSPITAL 1982

Folder No. _____ Research No. _____ — (1) — (2) — (3)

SURNAME _____ FIRST NAME _____ 1 (4)

Tested Before? Yes/No AGE ___ WEIGHT _____ (kg) R/S (7) ___ — (5) — (6)

SECTION I: TO BE FILLED OUT BY DOCTOR — (7)

Appearance of patient — (8)

Underweight	1	Overweight	3
Normal	2	Obese	4

Type of Diabetes: — (9)

Insulin-dependent	1
non-insulin dependent	2
Pancreatic	3

Known duration of Diabetes: — (10)

< 1 Year/s	1
1-3	2
3-5	3
5-10	4
> 10	5

No. of Hospital admissions (For control of diabetes) — (11)

None	1
1	2
2-3	3
More than 3	4

How many other admissions? — (12)

None	1
1	2
2-3	3
More than 3	4

Reasons: _____

_____

Form of treatment: (Type and dose) — (13)

Insulin	1	.....
Tablets	2	.....
Diet only	3	

Frequency of Visits: — (14)

1	Monthly
2	3 Monthly
3	4 Monthly
4	6 monthly

Standard of control: (mean of past year's blood glucose values) — (15)

≤ 8.5	Excellent	1
8.6-11	Average	2
11.1-13.9	Fair	3
≥ 14	Poor	4

(HbA1c _____) — (16) — (17) — (18)

Doctor's opinion on compliance: — (19)

Diet:	Good	1	Medications:	Good	1
	Average	2		Average	2
	Poor	3		Poor	3

— (20)

SECTION 1. (Contd.)

TO BE COMPLETED BY ALL PATIENTS

1. What education have you had?

Illiterate	1
Lower Primary	2
Higher Primary	3
High School	4
Matriculation	5
Technical	6
University	7

— (21)

2. Have you seen the films on diabetes? Yes ☐ No ☐ — (22)

3. Which films have you seen:

General diet	
Weight reduction diet	
Diet for insulin	
Insulin treatment	

How many times have you seen the films altogether?

Once	1
Twice	2
Three times	3
More than three times	4

— (23)

SECTION II:

4. What does "Diabetes" mean? (More than one answer may be given)

Too much sugar in the blood	
Too much sugar in the urine	
Too little sugar	
Not enough insulin in the body	
A type of blood disorder	

— (24)

5. Which of this list of complaints might a patient have at the start of their diabetes? (More than one answer may be given)

Thirst	
Genital itch	
Coughing	
Leg cramps	
Passing a lot of urine	
Boils	
Diarrhoea	
Chest pain	
Feeling ill	

— (25)

DEEL 1 : (Vervolg)

MOET ASSEBLIEF DEUR ALLE PASIËNTE INGEVUL WORD

1. Wat is u vlak van geleerdheid?

Ongeleerd	1
Onder Laerskool	2
Hoër Laerskool	3
Hoërskool	4
Matrikulasie	5
Tegnies	6
Universiteit	7

— (21)

2. Het u die skyfie-vertoning oor Diabetes gesien?

Ja ☐ Nee ☐

— (22)

3. Watter vertonings het u gesien?

"General diet"	
"Weight reduction diet"	
"Diet for insulin"	
"Insulin treatment"	

Hoeveel keer het u die vertonings altesame gesien?

Een keer	1
Twee keer	2
Drie keer	3
Meer as drie keer	4

— (23)

DEEL II:

4. Wat beteken "Diabetes" ? (suikersiekte)
(Meer as een antwoord mag gegee word)

Te veel suiker in die bloed	
Te veel suiker in die urien	
Te min suiker	
Nie genoeg insulien in die liggaam	
'n Soort bloedsiekte	

— (24)

5. Watter van dié lys klagtes mag 'n pasiënt hê in die begin van hulle suikersiekte? Meer as een antwoord mag gegee word.

Dors	
Jeuk in die private dele	
Hoes	
Beenkrampe	
Passeer baie water	
Absesse	
Diaree	
Borspyn	
Voel sleg	

— (25)

6. What does exercise or hard work do to the blood sugar?

Makes it go up	☐
Makes it go down	☐
Don't know	☐

— (26)

7. When during the day do you test your urine? (One answer)

Early morning (first) specimen	☐
Second urine specimen	☐
Before each meal	☐
Other times (specify)	☐

— (27)

8. What do you do if there is more sugar than usual in your urine? (More than one answer may be given)

Take more insulin/tablets	☐
Take less insulin/tablets	☐
Eat more	☐
Eat less	☐
Consult a doctor	☐
Exercise	☐
Nothing	☐

— (28)

9. Do you use a hot-water bottle? Yes ☐ No ☐

If you do, describe how you use it .....
.....
.....

— (29)

10. Do you know of any possible long-term complications of diabetes?

Yes ☐ No ☐

— (30)

If so, please state as many as you know. .....
.....
.....

11. Which of the factors listed below will improve your general health as a diabetic? (More than one answer may be given)

Taking good care of your feet	☐
Avoiding exercise	☐
Regular eye check-ups	☐
Following your diet	☐
Smoking	☐
Testing your urine regularly	☐
Gaining weight	☐
Learning more about your illness	☐

— (31)

6. Wat doen oefening of harde werk aan die bloedsuiker?

Verhoog dit	☐
Verlaag dit	☐
Weet nie	☐

— (26)

7. Wanneer gedurende die dag toets u u urien? (Een antwoord)

Vroeg in die oggend (eerste monster)	☐
Tweede monster van die dag	☐
Voor elke maaltyd	☐
Ander tye (meld asseblief)	☐

— (27)

8. Wat doen u as daar meer suiker as gewoonlik in u urien is? (Meer as een antwoord mag gegee word)

Neem meer insulien/tablette	☐
Neem minder insulien/tablette	☐
Eet meer	☐
Eet minder	☐
Vra 'n dokter	☐
Oefening	☐
Niks	☐

— (28)

9. Gebruik u 'n warmwatersak? Ja ☐ Nee ☐

Indien wel, beskryf hoe u dit gebruik .....

.....

.....

— (29)

10. Weet u van enige moontlike langtermyn komplikasies van suikersiekte?

Ja ☐ Nee ☐

Indien so, skryf asseblief neer soveel as wat u van weet

.....

.....

— (30)

11. Watter van die faktore in die onderstaande lys sal u algemene gesondheid as 'n diabeet verbeter? (Meer as een antwoord mag gegee word)

Pas u voete deeglik op	☐
Vermy oefening	☐
Gereelde oog ondersoeke	☐
Hou by u diëet	☐
Rook	☐
Toets u urien gereeld	☐
Tel gewig op	☐
Weet meer van u siekte	☐

— (31)

12. Have you been told to keep a record of your urine tests?

Yes ☐
No ☐

— (32)

13. Have you been told to look after your feet carefully?

Yes ☐
No ☐

— (33)

14. Do you test your urine? Yes ☐ No ☐

— (34)

15. How often do you test your urine? (One answer)

Never ☐
1-3 times a week ☐
Once a day ☐
Twice a day ☐
3 or more times a day ☐

— (35)

16. Do you keep a record of your urine testing?

Yes ☐
No ☐

— (36)

17. Has the special Diabetic Sister explained Diabetes and urine testing to you?

Yes ☐
No ☐

— (37)

Please turn over for next section.

12. Is u ooit gevra om 'n rekord te hou van u uriene toetse?

Ja ☐
Nee ☐ — (32)

13. Is u ooit gesê om u voete deeglik op te pas?

Ja ☐
Nee ☐ — (33)

14. Toets u wel u uriene?

Ja ☐
Nee ☐ — (34)

15. Hoe dikwels toets u u uriene?

Nooit ☐
1-3 keer per week ☐
Een keer per dag ☐ — (35)
Twee keer per dag ☐
3 of meer keer per dag ☐

16. Hoe u 'n rekord van u uriene toetse?

Ja ☐
Nee ☐ — (36)

17. Het die spesiale Diabetiese Suster vir u diabetes en uriene toetse verduidelik?

Ja ☐
Nee ☐ — (37)

Blaai asseblief om vir volgende deel

SECTION III

TO BE COMPLETED BY ALL PATIENTS

1. What kind of diet are you following?

None ☐
Weight reducing ☐
Diabetic ☐
Other ☐

— (38)

2. How many calories are you allowed each day?

1000 ☐ 1600 ☐ 2400 ☐
1200 ☐ 1800 ☐ 2600 ☐ Not sure ☐
1400 ☐ 2000 ☐ 2800 ☐

— (39)

3. Have you been told what a carbohydrate (starch) exchange or "count" is?

Yes ☐
No ☐

— (40)

If so, explain in your own words .....

.....

4. If you know, write down the total number of exchanges (counts) that you are allowed each day in your diet. .....

— (41)

5. Tick off any foods in the list below which contain a lot of carbohydrate (starch or sugar)

meat ☐ porridge ☐ bread ☐
bean soup ☐ margarine ☐ milk ☐
oil ☐ eggs ☐ rice ☐
fruit ☐ potatoes ☐ cream ☐

— (42)

6. Tick off any foods in the list below which are allowed freely in a diabetic or weight-reduction diet.

Oros ☐ canned fruit ☐ beer ☐
cabbage ☐ honey ☐ chutney ☐
glucose sweets ☐ soda water ☐ lettuce ☐
tomatoes ☐ condensed milk ☐ carrots ☐
clear soup ☐ yoghurt ☐ cauliflower ☐

— (43)

DEEL III

MOET DEUR ALLE PASIëNTE INGEVUL WORD

1. Watter soort dieet volg u?

Geen dieet	☐
Om gewig te verloor	☐
Diabeties	☐
Ander	☐

— (38)

2. Hoeveel kalorie mag u elke dag inneem?

1000	☐	1600	☐	2400	☐		
1200	☐	1800	☐	2600	☐	nie seker	☐
1400	☐	2000	☐	2800	☐	nie	

— (39)

3. Is u vertel wat 'n koolhidraat (stysel) omruiling is?

Ja	☐
Nee	☐

— (40)

Indien wel, verduidelik in u eie woorde .....

.....

4. Indien u weet, skryf neer die totale nommer van omruilings wat u elke dag volgens u dieet mag neem ..... — (41)

5. Merk enige kossoorte in die onderstaande lys wat baie koolhidraat (stysel of suiker) bevat.

vleis	☐	pap	☐	brood	☐
boontjiesop	☐	margarien	☐	melk	☐
olie	☐	eiers	☐	rys	☐
vrugte	☐	aartappels	☐	room	☐

— (42)

6. Merk enige kossoorte in die onderstaande lys wat u vrylik in 'n diabetiese of gewigsverlorende dieet mag gebruik.

oros	☐	ingelegte vrugte	☐	bier	☐
kool	☐	heuning	☐	blatjang	☐
glukose lekkers	☐	sodawater	☐	blaarslaai	☐
tamaties	☐	gekondenseerde melk	☐	geelwortels	☐
helder sop	☐	yoghurt	☐	blomkool	☐

— (43)

7. If you are on a weight reduction diet have you been told why it is good for you to lose weight ?

N/A ☐
Yes ☐
No ☐

— (44)

If so, explain in your own words.

.....

.....

.....

8. How much is allowed for one serving of the following?

.e.g..½.cup.....	Porridge
.....1.....	Apple
.....	Rice
.....	Grapes
.....	Potato
.....	Thick soup

— (45)

9. Why do you think following a correct diet is important for diabetic people?

.....

.....

.....

— (46)

10. Have you been told to see a dietitian about your diet?

Yes ☐
No ☐

— (47)

11. How many times have you been to see her?

Never ☐
Once ☐
Twice ☐
Three times ☐
More ☐

— (48)

12. Has the dietitian given you a diet booklet?

Yes ☐
No ☐

— (49)

7. As u op 'n gewigverlorende dieet is, is u vertel hoekom dit goed vir u is om gewig te verloor?

n.v.t. ☐
Ja ☐
Nee ☐

— (44)

Indien so, verduidelik in u eie woorde.

.....

.....

.....

8. Hoeveel word toegelaat as 'n enkele porsie van die volgende?

bv. ½ koppie.... pap
...een..... appel
..... rys
..... druiwe
..... aartappels
..... diksop

— (45)

9. Waarom dink u dat 'n korrekte dieet belangrik is vir diabete?

.....

.....

.....

— (46)

10. Is u ooit na die dieëtkundige gestuur oor u dieet?

Ja ☐
Nee ☐

— (47)

11. Hoe dikwels het u haar al gesien?

Nooit ☐
Een keer ☐
Twee keer ☐
Drie keer ☐
meer as drie keer ☐

— (48)

12. Het die dieëtkundige vir u 'n dieëtboekie gegee?

Ja ☐
Nee ☐

— (49)

13. Have you been told what diet to follow? — (50)

Yes ☐
No ☐

14. Have you been told that you must not use sugar? — (51)

Yes ☐
No ☐

15. Have you been told you may use saccharin? — (52)

Yes ☐
No ☐

16. Which kind of milk have you been told to drink? — (53)

Condensed milk ☐ Ordinary cow's milk ☐
Skimmed milk ☐ None in particular ☐

17. Have you been told to use oil or "soft" margarine for cooking or eating? — (54)

Yes ☐
No ☐

18. Have you been told which cold drinks you may have? — (55)

Yes ☐
No ☐

19. Have you been told which foods contain carbohydrate (starch and sugar)? — (56)

Yes ☐
No ☐

20. Have you been given a list of foods which are unrestricted (allowed freely) in your diet? — (57)

Yes ☐
No ☐

21. Have you been told how many carbohydrate (starch) exchanges you may have each day in your diet? — (58)

Yes ☐
No ☐

13. Is u vertel watter dieet u mag volg?

Ja ☐
Nee ☐ — (50)

14. Is u vertel dat u nie suiker mag neem nie?

Ja ☐
Nee ☐ — (51)

15. Is u vertel dat u sakkarien mag gebruik?

Ja ☐
Nee ☐ — (52)

16. Watter soort melk is u vertel om te drink?

Gekondenseerde melk ☐ Gewone beesmelk ☐
afgeroomde melk ☐ niks besonders nie ☐ — (53)

17. Is u vertel om olie of sagte margarien vir kook of eet te gebruik?

Ja ☐
Nee ☐ — (54)

18. Is u vertel watter koeldranke u mag gebruik ?

Ja ☐
Nee ☐ — (55)

19. Is u vertel watter soort kos bevat koolhidraat (stysel en suiker) ?

Ja ☐
Nee ☐ — (56)

20. Is u 'n lys gegee van kossoorte wat u mag vrylik gebruik in u dieet?

Ja ☐
Nee ☐ — (57)

21. Is u vertel hoeveel styselomruilings u elke dag volgens u dieet mag neem?

Ja ☐
Nee ☐ — (58)

22. What do you take with your tea or coffee?

Nothing	☐	Skim milk	☐
Non-dairy creamer	☐	Condensed milk	☐
Cow's milk	☐		

— (59)

23. Which of the following do you take in your tea or coffee?

Honey	☐	Saccharin	☐
Sugar	☐	None	☐

— (60)

24. What kinds of jams and marmalades do you take?

Ordinary	☐
Diabetic	☐
None	☐

— (61)

25. Which of the following do you use for cooking?

Cooking fat (e.g. Holsum)	☐	Tub margarine (e.g. Floro)	☐
Cooking oil	☐	Wrapped marg.(Blossom)	☐
		Butter	☐

— (62)

26. Which of the following do you use for your bread?

Dripping	☐	Wrapped marg.	☐
Tub marg.	☐	Butter	☐
		Other	☐

— (63)

27. Which of the following drinks do you take?

		How much?	How often?
dry wine	☐	.....	.....
beer	☐	.....	.....
sweet wine	☐	.....	.....
sherry	☐	.....	.....
other (specify)	☐	.....	.....
Don't drink	☐		

— (64)

28. What kind of bread do you use?

White	☐
Brown	☐
Wholewheat	☐
Crushed wheat	☐

— (65)

22. Wat voeg u by u tee of koffie?

Niks	☐	Afgeroomde melk	☐
Nie-suiwel roomer	☐	Gekondenseerde melk	☐
Beesmelk	☐		

— (59)

23. Watter van die volgende neem u in u tee of koffie?

heuning	☐	sakkarien	☐
suiker	☐	geen	☐

— (60)

24. Watter soorte konfyt of marmalade gebruik u?

gewone	☐
diabeties	☐
geen	☐

— (61)

25. Watter van die volgende gebruik u om mee te kook?

kookvet (b.v. Holsum)	☐	margarien in 'n bakkie (b.v. Floro)	☐
botter	☐		
kookolie	☐	omgevoude margarien (b.v. blossom)	☐

— (62)

26. Watter van die volgende gebruik u op u brood?

vet	☐	omgevoude margarien	☐
bakkie margarien	☐	botter	☐
		ander	☐

— (63)

27. Watter van die volgende drankies gebruik u?

		Hoeveel	Hoe dikwels
droëwyn	☐	.....	.....
bier	☐	.....	.....
soet wyn	☐	.....	.....
sjerrie	☐	.....	.....
ander (meld asb)		.....	.....
drink nie	☐		

— (64)

28. Watter soort brood gebruik u?

wit	☐
bruin	☐
volkoring	☐
stampkoring	☐

— (65)

29. Describe in detail what you had to eat or drink yesterday.
Write down the size of your helpings if possible.

Early morning .....

.....

Breakfast .....

.....

.....

Mid-morning .....

Lunch .....

.....

.....

Mid-afternoon .....

Supper .....

.....

.....

.....

Late night .....

.....

Was this a usual day for your diet? Yes ☐ No ☐

If not, explain .....

.....

29. Meld in volle besonderhede wat u gister gehad het om te eet en drink. Skrywe neer die groote van die porsies indien moontlik.

Vroegoggend .....

.....

Ontbyt .....

.....

.....

Middeloggend .....

Middagete .....

.....

.....

Middagtee .....

Aandete .....

.....

.....

.....

Laataand .....

.....

Was dit 'n gewone dag vir u dieet? Ja ☐ Nee ☐

Indien nie, verduidelik .....

.....

SECTION IV

ONLY TO BE COMPLETED BY PATIENTS ON TABLETS TO CONTROL THEIR SUGAR

1. Do you know the name(s) of your tablets?

Yes ☐
No ☐ — (67)

2. If so, what tablets do you use? .....

.....

3. Do you know the dose you are taking?

Yes ☐
No ☐ — (68)

What is it? .....

4. Is this what your doctor told you to take?

Yes ☐
No ☐ — (69)

5. Do you have tablets left over at the end of the month? (One answer)

Never ☐
Run short of tablets ☐
Sometimes have tablets over ☐ — (70)
Always have tablets over ☐

DEEL IV

MOET SLEGS INGEVUL WORD DEUR PASIëNTE OP TABLETTE OM HULLE SUIKER TE BEHEER

1. Ken u die name van u tablette?

Ja ☐
Nee ☐ — (67)

2. Indien so, watter tablette gebruik u? .....

.....

3. Ken u die dosis wat u neem?

Ja ☐
Nee ☐ — (68)

Wat is dit? .....

4. Is dit wat u dokter voorgeskryf het?

Ja ☐
Nee ☐ — (69)

5. Het u tablette oor teen die einde van die maand?

nooit ☐
te min tablette ☐
somtyds het tablette oor ☐ — (70)
het altyd tablette oor ☐

SECTION V

— (1) — (2) — (3)

2 (4)

ONLY TO BE COMPLETED BY PATIENTS TAKING INSULIN

1. Has the special Diabetic Sister taught you about your insulin injections?

   Yes ☐
   No ☐ — (5)

2. Do you know what type of insulin you use?

   Yes ☐
   No ☐ — (6)

   What type do you use? .....

3. What strength insulin do you use?

   40 units per ml. ☐
   80 units per ml. ☐ — (7)

4. How often do you take insulin?

   Once a day ☐
   Twice a day ☐
   Three times a day ☐
   More ☐ — (8)

5. What dose of insulin do you take? .....

6. Do you change your insulin dose sometimes?

   Yes ☐
   No ☐ — (9)

7. If you answered "yes" to question 6, which of the following would make you take more insulin. (More than 1 answer may be given)

   No sugar in the urine ☐
   Ketones in the urine ☐
   Too much sugar in the urine ☐
   Illness or infection ☐
   Hypoglycaemic attack (low sugar) ☐
   Missing a meal ☐
   Exercise or hard physical work ☐
   big meal ☐ — (10)

DEEL V — (1) — (2) — (3)

MOET NET INGEVUL WORD DEUR PASIëNTE WAT INSULIEN GEBRUIK. 2 (4)

1. Het die spesiale Diabetiese Suster u geleer hoe om u insulien te gebruik? — (5)

Ja	☐
Nee	☐

2. Weet u watter soort insulien u gebruik? — (6)

Ja	☐
Nee	☐

Watter soort gebruik u? .....

3. Watter sterkte insulien gebruik u? — (7)

40 eenhede per ml.	☐
80 eenhede per ml.	☐

4. Hoe dikwels neem u insulien? — (8)

een keer 'n dag	☐
twee keer 'n dag	☐
drie keer 'n dag	☐
meer	☐

5. Watter dosis insulien neem u? .....

6. Verander u u insulien dosis somtyds? — (9)

Ja	☐
Nee	☐

7. Indien u "ja" vir vraag 6 gemerk het, watter van die volgende faktore sou u u insuliendosis laat <u>vermeerder</u>? (Meer as een antwoord mag gegee word) — (10)

geen suiker in u uriene	☐
ketone in u uriene	☐
te veel suiker in u uriene	☐
siekte of infeksie	☐
hipoglisemiese aanval (lae suiker)	☐
maaltyd oorslaan	☐
oefening of harde liggaamlike werk	☐
groot maaltyd	☐

8. Which of the following would make you take <u>less</u> insulin?

(More than one answer may be given)

No sugar in the urine	☐
Ketones in the urine	☐
Too much sugar in the urine	☐
Illness or infection	☐
Hypoglycaemia attack (Low sugar)	☐
Missing a meal	☐
Exercise or hard physical work	☐
Big meal	☐

— (11)

9. What would one expect to feel during a hypoglycaemic attack?

(More than one answer may be given)

Sweating	☐
Headache	☐
Vomiting	☐
Hunger	☐
Palpitations	☐
Back pain	☐
Tremor	☐
Blurring of vision	☐
Diarrhoea	☐
Coma	☐
Cough	☐

— (12)

10. Which of these could make your blood sugar fall too low and cause a hypoglycaemic attack? (More than 1 answer may be given)

Too much insulin	☐
Too little insulin	☐
Missing a meal	☐
Not eating enough	☐
Over-eating	☐
Exercise	☐
Infection/Illness	☐
Too much alcohol	☐

— (13)

8. Watter van die volgende sou u <u>minder</u> insulien laat neem? (Meer as een antwoord mag gegee word)

geen suiker in u uriene	
ketone in u uriene	
te veel suiker in uriene	
siekte of infeksie	
hypoglisemiese aanval (lae suiker)	
maaltyd oorslaan	
oefening of harde liggaamlike werk	
groot maaltyd	

— (11)

9. Wat sou 'n mens verwag om te voel gedurende 'n hipoglisemiese (lae suiker) aanval? (Meer as een antwoord mag gegee word)

sweet	
hoofpyn	
braking	
honger	
hartklopping	
rugpyn	
bewerigheid	
steuring van visie	
diarree	
koma	
hoes	

— (12)

10. Watter van die onderstaande faktore mag u bloedsuiker te laag laat val en 'n hipoglisemiese aanval veroorsaak? (Meer as een antwoord mag gegee word)

te veel insulien	
te min insulien	
maaltyd oorslaan	
nie genoeg eet nie	
te veel eet	
oefening	
infeksie/siekte	
oormatige alkohol inname	

— (13)

11. How would you treat a hypoglycaemic attack? (More than 1 answer may be given)

Take more insulin	☐
Eat a sweet	☐
Have a meal	☐
Take less insulin	☐

— (14)

12. How long does the effect of one injection of your type/s of insulin last in the body .....

..... — (15)

13. How would you prevent a hypoglycaemic attack occuring again? (More than one answer may be given)

Eat regularly	☐
Take more insulin	☐
Eat before exercise	☐
Take less insulin	☐
Eat more sweets	☐

— (16)

14. What do you do if ketones appear in your urine? (Not for patients who use "testape" only)

Take more insulin	☐
Take less insulin	☐
Eat more	☐
Eat less	☐
Consult a doctor	☐
Exercise	☐
Do nothing	☐

— (17)

15. FOR THOSE PATIENTS WHO USE <u>DISPOSABLE SYRINGES</u>.

(a) Do you wash your syringe after use? Yes ☐ No ☐ — (18)

(b) How and where do you keep your syringe between injections?

.....

..... — (19)

(c) How many times do you use the same syringe?

.....

11. Hoe sou u 'n hipoglisemiese aanval behandel?
(Meer as een antwoord mag gegee word)

neem meer insulien	☐
eet 'n lekker	☐
eet 'n maaltyd	☐
verminder u insulien dosis	☐

— (14)

12. Hoe lank duur die uitwerking van een inspuiting van u tipe insulien in die liggaam?

— (15)

.....

13. Hoe sou u verhoed dat 'n hipoglisemiese aanval gebeur?
(Meer as een antwoord mag gegee word)

eet gereeld	☐
neem meer insulien	☐
eet voor oefening	☐
neem minder insulien	☐
eet meer lekkers	☐

— (16)

14. Hoe sou u reageer indien ketone in u uriene verskyn?
(Nie vir pasiënte wat slegs "testape" gebruik nie)

neem meer insulien	☐
neem minder insulien	☐
eet meer	☐
eet minder	☐
vra 'n dokter	☐
oefen	☐
niks	☐

— (17)

15. ALLEENLIK VIR PASIëNTE WAT <u>WEGDOENBARE SPUITE</u> GEBRUIK?

(a) Spoel u u spuit uit na gebruik? Ja ☐ Nee ☐

(b) Hoe en waar stoor u u spuit?

— (18)

.....

.....

(c) Hoeveel keer gebruik u dieselfde spuit?

— (19)

.....

16. FOR THOSE PATIENTS WHO USE GLASS SYRINGES.

(a) Do you sterilise your syringe by boiling in water

Yes ☐
No ☐ — (18)

If not, how do you sterilise it? .....

.....

(b) How often do you sterilise your syringe? .....

.....

(c) Do you keep your syringe in spirits?

Yes ☐
No ☐ — (19)

If not, how do you store it? .....

.....

17. Your daily injection(s) should be given (one answer)

Into the skin ☐
Under the skin ☐
Into a muscle ☐ — (20)
Into a vein ☐

18. Where do you give your injections?

Thigh ☐
Stomach ☐
Buttock ☐ — (21)
Arm ☐

19. Do you use a slightly different place for your injection every day?

Yes ☐
No ☐ — (22)

16. ALLEENLIK VIR PASIëNTE WAT <u>GLASSPUITE</u> GEBRUIK

(a) Steriliseer u u spuit deur dit in water te kook? — (18)

Ja ☐
Nee ☐

Indien nie, hoe steriliseer u dit? .....

.....

(b) Hoe dikwels steriliseer u dit? ..... — (19)

.....

(c) Stoor u u spuit in spiritus?

Ja ☐
Nee ☐

Indien nie, hoe stoor u dit? .....

.....

17. u Daaglikse inspuiting(s) behoort ingespuit te word soos volg. (een antwoord) — (20)

in die vel ☐
net onder die vel ☐
in 'n spier ☐
in 'n aar ☐

18. Waar spuit u uself in? (Meer as een antwoord mag gegee word) — (21)

dy ☐
buik (maag) ☐
boude ☐
arm ☐

19. Gebruik u 'n verskillende plek elke dag vir u insulien inspuiting? — (22)

Ja ☐
Nee ☐

ENCLOSURE II

POINTS FOR GOOD HEALTH

1. Take very good care of your feet - keep them dry and cut toenails carefully. Wear comfortable shoes and socks. Clean and examine your feet and toes every day. Report any cuts or injuries to your doctor. Avoid hotwater bottles, unless wrapped in a thick towel or flannel.

2. Have your eyes checked yearly or sooner if you notice any change in your eyesight.

3. When going away even for a few days, be sure to have your supply of tablets or insulin and syringes, and also to continue with your diet.

4. Remember to test your urine at least once a day, and to empty your bladder 20 minutes before passing urine for the test. It is useful to keep a record of your urine tests for your doctor to see.

5. It is best to be slightly <u>under</u> your ideal weight for your height, and to keep your weight at this level. Ask the dietitian for advice about this.

6. Always carry some form of identification i.e. name, address and the fact that you are a diabetic. A medic-alert bracelet is obtainable - ask the sister to help you fill in the form.

7. Try to be active and walk about half an hour every day in the fresh air.

THE RIGHT WAY TO EAT

1. Eat regular meals. Never miss a meal. Have snacks as allowed.

2. Eat some meat, fish, chicken, cheese or eggs as allowed, with every meal or snack. Choose fat-free foods if you are overweight. You may use plain yoghurt or peanut butter or dried beans, peas or lentils instead of meat.

3. Learn to count the total number of helpings of starchy food that you can have every day from the lists of exchanges for bread, cereals (rice, porridge, etc.) fruit and starchy vegetables as given in your diet booklet. You may exchange a medium apple for a slice of bread for example.

4. Try to have vegetables from the free list, raw and cooked, every day.

5. Use soft margarine in a tub, rather than butter or margarine wrapped in paper. Use oil for cooking if you are not overweight.

6. Use your allowance of milk for breakfast cereals, or to drink during the day. Skimmed milk is best. Never use non-dairy creamers or condensed milk.

7. <u>Never</u> take foods or drinks which contain sugar, unless you have been told to use them in an emergency such as a "hypo attack". This means that you must not eat cakes, sweet biscuits, ordinary canned fruit, jelly custard, ordinary jam, marmalade, honey, syrup, thick sauces etc. You may use saccharin for sweetening food if necessary. Ask the dietitian about special "diet" foods, before you buy them!

REëLS VIR GOEIE GESONDHEID

1. Pas u voete deeglik op - hou hulle droog and sny toonnaels versigtig. Dra gemaklike skoene en sokkies. Was en ondersoek u voete en tone elke dag. Rapporteer snye of beserings aan u dokter. Moenie warmwatersakke gebruik nie, tensy toegedraai in n' handdoek.

2. Laat u oë toets elke jaar asook vroeër as u enige verandering opmerk in u oë.

3. As u weggaan al is dit net vir n' paar dae, maak seker dat u u regte hoeveelheid pille neem of genoeg insulien en spuite en ook om aan te gaan met u dieët.

4. Onthou om u urine tenminste een keer per dag te toets en om u blaas te ledig 20 minute voordat u die urine wil toets. Dit is handig om n' rekord te hou van u urine toetse sodat u dokter kan sien.

5. Onthou dit is beter om effens minder te weeg as u ideale gewig vir u lengte en probeer om u gewig so te hou. Vra die dieëtkundige vir advies omtrent u gewig.

6. Sorg altyd dat u n' vorm van identifikasie by u het, byvoorbeeld u naam, adres asook dat u n' diabeet is. n' Medic Alert armband is verkrygbaar - vra die suster by die hospitaal om die vorm vir u in te vul.

7. Probeer aktief bly en stap byvoorbeeld vir omtrent half uur in die vars lug elke dag.

DIE KORREKTE EETGEWOONTES.

1. Eet gereelde maaltye. Moenie n' maaltyd oorslaan nie. Tussen happies word toegelaat.

2. Eet vleis, vis, hoender, kaas of eiers soos toegelaat, met elke maaltyd of happie. Eet vet-vrye kossoorte as u oorgewig is. u Mag gewone joghurt of grondboontjiebotter of droë boontjies, ertjies of lensies gebruik in plaas van vleis.

3. u Moet leer om die totale hoeveelheid porsies styselagtige kossoorte dat u mag neem van die lys van omruilings vir brood, graansoorte (rys, pap ens) vrugte en styselagtige groente soos aangegee in u dieëtboekie te tel. u Mag byvoorbeeld n' medium groote appel omruil vir een sny brood.

4. Probeer om elke dag groente wat vrylik toegelaat word te eet rou en gekook.

5. Gebruik liefs die margarien in n' bakkie in plaas van botter of margarien toegedraai in papier of omhulsel. As u nie oorgewig is nie, gebruik dan olie om mee te kook.

6. Gebruik u toegelate hoeveelheid melk vir ontbyt graansoorte of om te drink gedurende die dag. Afgeroomde melk is die beste. Moet asseblief nooit nie-suiwel roomers of gekondenseerde melk gebruik nie.

7. Moet nooit kossoorte of drankies neem wat suiker bevat nie, tensy u aangesê is om hulle net in n' noodgeval te neem byvoorbeeld n'"hipo aanval". Dit beteken dat u nie koek, soet koekies, ordinêre ingemaakte vrugte, jellie, vla, ordinêre konfyt, marmalade, heuning, stroop, dik souse ensovoorts mag eet nie. u Mag sakkarien gebruik om kos mee te versoet. Vra die dieëtkundige omtrent spesiale "dieët kosse" voordat u hulle aankoop.

ENCLOSURE III

INSTRUCTIONS TO PATIENTS

Please write down EVERYTHING you eat, drink or nibble each day, from the time you get up until the time you go to sleep. We need to know all the details; for example -

1. How many spoons of food you have on your plate and what size they are. (See bottom of page).

2. How big your glass or cup is. (Small, medium or large).

3. How big the slices or pieces of meat, fish, cheese, fruit or vegetables are. (Small, medium or large).

4. How the food has been cooked, whether boiled, steamed, baked, stewed, fried (in oil or fat), roasted or grilled.

5. What you eat with your food and how much - tomato sauce, chutney, pickles, salad dressing, mayonnaise, gravy made with flour or Bisto.

6. What kind of meat and fish you eat; for example, brisket, flank, mince, mutton, stockfish, snoek, pilchards and so on.

7. What kind of cheese; for example, sweetmilk, cheddar, cottage.

8. What kind of bread and how thick the slice is.

9. Whether you use butter, margarine or other spreads on your bread.

10. What kind of pudding you have , with custard, cream, or spmething else.

11. What kind of biscuits, sweets and cake you have. (Sometimes!)

ABBREVIATIONS: t = teaspoon
d = dessertspoon or pudding spoon
T = Tablespoon
S = Serving spoon or kitchen spoon

DAY: _____

EARLY MORNING:.....

.....

BREAKFAST:.....

.....

.....

.....

.....

MID-MORNING:.....

.....

LUNCH:.....

.....

.....

.....

.....

.....

.....

MID-AFTERNOON:.....

.....

SUPPER:.....

.....

.....

.....

.....

.....

.....

LATE-NIGHT:.....

.....

.....

.....

.....

.....

DIETARY ANALYSIS

Research No: _____

	Amt(g)	Frequ.	Amt/wk	CALS	PROT	FAT	C-O	FIBRE
A. BREAD AND CEREALS								
Oats								
Mieliemeal								
Weetbix								
Maltabella								
Pronutro								
Corn Flakes								
Other:								
Sugar added								
Samp								
Samp and Beans (3:1 or 5:1)								
Bread (white)								
Bread (brown)								
Bread (whole, crushed wheat)								
Butter/Margarine								
Rolls								
Cream crackers								
Provita								
Rice								
Spaghetti (boiled)								
Spaghetti (canned in sauce)								
Macaroni								
Cheese sauce								
Noodles								
B. SOUPS AND GRAVY								
Soup (pkt)								
Soup (split-pea)								
Soup (bean)								
Soup (barley)								

	Amt(g)	Freq	Amt/wk	CALS	PROT	FAT	CHO	FIBRE
Soup (vegetable)								
Fat/oil added								
Gravy (Bisto, flour)								
Other:								
MEAT, FISH, EGGS, etc.								
Beef (steak)								
Beef (stew)								
Beef (roast, grilled)								
Beef (corned, tinned)								
Chicken								
Liver								
Kidneys								
Mince								
Tripe, lungs								
Brains								
Mutton (chops)								
Mutton (stew)								
Mutton (roast)								
Pork								
Bacon								
Polony								
Cold meats								
Sausage (beef, pork)								
Sausage (vienna)								
Sausage rolls								
Meat pies								
Veal								
Fish (steamed)								
Fish (fried)								
Haddock								

	Amt(g)	Freq	Amt/wk	CALS	PROT	FAT	CHO	FIBRE
Fillchamps								
Other:								
Cheese (cheddar)								
Cheese (skim milk)								
Cheese (Cottage)								
Egg (boiled)								
Egg (fried)								
Egg (scrambled)								
Soya products								
Fat/oil added								
D. VEGETABLES								
Beans (baked and sauce)								
Beans (green)								
Beetroot								
Broccoli								
Cabbage (raw, cooked)								
Carrots (raw, cooked)								
Cauliflower								
Mielies								
Sweetcorn								
Onion								
Peas (raw, cooked)								
Potato(boiled)								
Potato (mashed)								
Potato (chips, roast)								
Potato (baked in skin)								
Pumpkin								
Radish								
Spinach								
Squash								

	Amt(g)	Frequ	Amt/wk	CALS	PROT	FAT	CHO	FIBRE
Sweetpotato								
Turnips								
Lettuce								
Tomato (raw, cooked)								
Cucumber								
Avocado								
Coleslaw								
Potato salad								
Fat/oil added								
Sugar added								
Mixed veg.								
Other:								
E. FRUIT								
Apple								
Apricot								
Banana								
Canned fruit								
Fruit salad								
Grapes								
Grapefruit								
Guavas								
Melon								
Naartjie								
Orange								
Pawpaw								
Peach								
Pear								
Pineapple								
Plum								
Dried Fruit								

	Amt(g)	Frequ	Amt/wk	CALS	PROT	FAT	CHO	FIBRE
Stewed fruit								
Other:								
MILK AND BEVERAGES								
Milk (full-cream)								
Milk (skimmed)								
Milk (condensed)								
Milk (evaporated)								
Buttermilk								
Yoghurt (plain)								
Yoghurt (fruit)								
Cream								
Milo (powder)								
Cocoa (powder)								
Sugar (in tea etc.)								
Non-dairy creamer								
Fruit juice								
Cordial								
Cold-drinks								
Beer								
Wine								
Other alcohol:								
MISCELLANEOUS								
Peanutbutter								
Honey								
Jam								
Syrup								
Mayonnaise								
Chutney								
Tomato sauce								

	Amt(g)	Frequ.	Amt/wk	CALS	PROT	FAT	CHO	FIBRE
Salad dressing								
Custard								
Jelly								
Icecream								
Steamed pudding								
Instant pudding								
Trifle								
Sago pudding								
Peanuts								
Chips (pkt)								
Chocolate								
Sweets								
Biscuits (asstd)								
Doughnuts								
Scones								
Cake								
Pies (fruit)								
Other:								

ENCLOSURE IV

MR KOLBE

1. Change all bread to WHOLEWHEAT. Have 4 SLICES a day.

2. Put MARGARINE only on HALF bread e.g. on one slice of sandwich.

3. Have 1 teacup WEETBIX every morning.

4. Add 2 TEASPOONS BRAN to Weetbix every morning.

5. Change cream crackers to PROVITA. Have 3 STRIPS a day.

6. Have 2 TABLESPOONS BAKED BEANS OR PEAS every night.

7. Have 6 PRUNES every day.

8. Have all POTATO WITH SKIN (eat the skin).

9. Keep to rest of diet as in orange booklet.

e.g. BREAKFAST: 1 teacup Weetbix and 2 teaspoons bran
1 slice bread

TEA: 6 prunes

LUNCH: 2 slices bread

TEA: 3 provita, or 1 fruit

SUPPER: 2 Tablespoons baked beans or peas
1 potato with skin, or 1 fruit

LATE NIGHT: 1 slice bread

MRS BESTER

1. Change all bread to WHOLEWHEAT. Have 2 slices a day. Use MARGARINE only on half bread (on one half of sandwich).

2. Have 1 TEACUP WEETBIX every morning.

3. Add 2 TEASPOONS BRAN to Weetbix every morning.

4. Change all POTATO to baked or boiled with skin (eat the skin). Have ONE every day.

5. Have 2 TABLESPOONS BAKED BEANS OR PEAS every day.

6. Have 6 PRUNES every night.

7. Change all milk to SKIMMED MILK. Have $\frac{1}{4}$ LITRE every day.

8. Keep to rest of diet as in diet booklet.
   7 STARCHES a day.

   e.g. BREAKFAST: 1 teacup Weetbix and 2 teaspoons bran

   LUNCH: 2 slices wholewheat bread

   SUPPER: 1 potato with skin
   2 Tablespoons baked beans or peas
   2 Tablespoons rice

   LATE NIGHT: 6 prunes

9. Leave out all DOUGHNUTS, SWEETS, CAKES AND ROLLS.

ENCLOSURE V

URINE TEST CHART (Indicate +, ++, +++)

NAME: .....

DATE COMMENCED: ..... WEIGHT: ..... (kg)

DAY	BEFORE BREAKFAST	BEFORE LUNCH	BEFORE SUPPER
0			
1			
2			
3			
4			
5			
6			
7			
8			
9			
10			
11			
12			
13			
14			

DATE CONCLUDED: 20/8/80 WEIGHT: 56.0 (kg)

URINE TEST CHART (Indicate +, ++, +++)

NAME: Mr. R. Smith

DATE COMMENCED: 2/18/80 WEIGHT: 86.0 (kg)

DAY	BEFORE BREAKFAST	BEFORE LUNCH	BEFORE SUPPER
0	0		
1			
2			
3			
4			
5			
6			
7			
8			
9			
10			
11			
12			
13			
14	0		

Next urine → r. 85 am thurs.

DATE CONCLUDED: ..... WEIGHT: 85.5 (kg)